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VISIT...

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Surface anatomy of the chest

Trachea

- From 6th cervical vertebra to 4th thoracic vertebra (angle of Lewis).
- 10 cm length (upper ½ in the neck and lower ½ in the chest).

Lung

- Apex.
- Anterior border:
- Inferior border.
- Posterior border.

a. apex

Represented by curved line, a point 3 cm above the medial ½ of the clavicle.

b. Anterior border

Represented by a line drawn downwards and medially from the sterno-clavicular joint to the sternal angle near the midline, then:

- In the right lung: it extends vertically downwards to the 6th sternocostal junction.
- In the left lung: it extends vertically downwards to the level of the 4th costal cartilage, where it curves laterally for 3.5 cm and then downwards and medially to reach the 6th costal cartilage 4 cm from the midline.

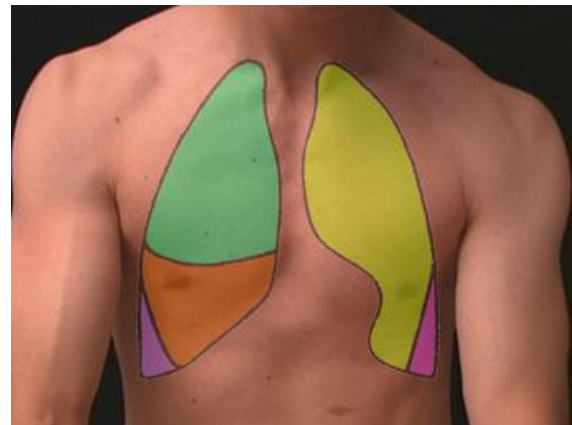
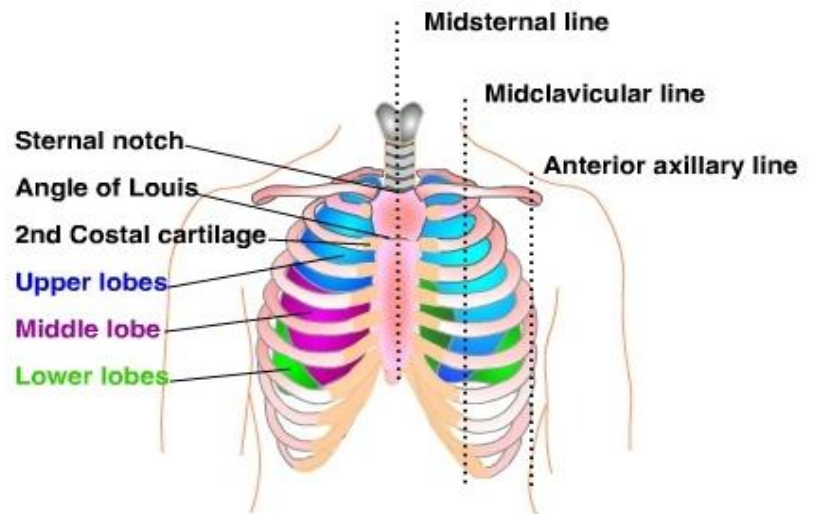
c. Inferior border

Represented by a line drawn laterally and backwards cutting the following ribs:

- 6th rib, at the midclavicular line.
- 8th rib, at the midaxillary line.
- 10th rib, at the scapular line.

d. Posterior border

Represented by a vertical line drawn from the posterior end of the inferior border up to the apex of the lung.



N.B. ☺

- Surface anatomy of the pleura: the same as lung by add 2 ribs over the inferior border of the lung.

Lung fissures

a. Oblique fissure: (both lungs).

النهاية	المنتصف	البداية
MCL → 6th costo-chondral junction	Extends obliquely downward and forward along the course of 6th rib (MAL)	T3 posteriorly.

b. Transverse fissure:

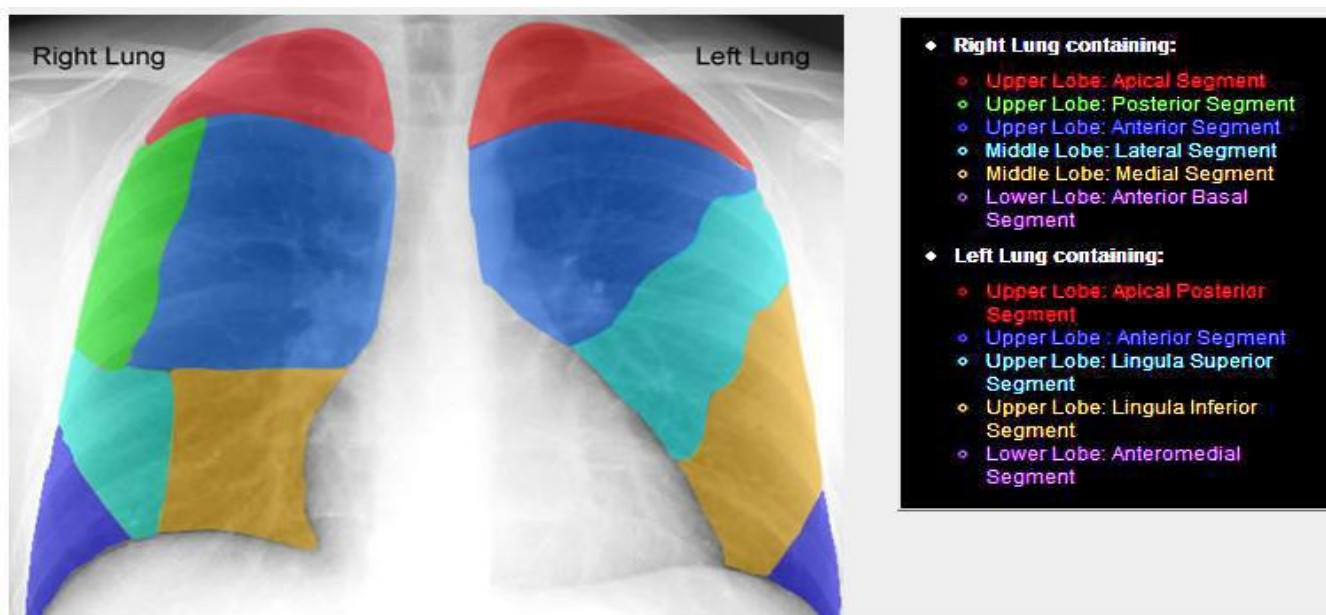
النهاية	البداية
6th rib at the MAL.	Sternal end at 4th costo-chondral junction laterally

Segments of the lung**A. Right lung (3 lobes)**

Upper (3 segments)	Middle (2 segments)	Lower (5 segments)
1. Apical. 2. Anterior. 3. Posterior.	1. Medial. 2. Lateral.	1. Superior. 2. Anterior. 3. Posterior. 4. Medial. 5. Lateral.

B. Left lung (2 lobes)

Upper lobe	Lower (4 segments).
• Proper (2 segments) 1. Apico-posterior. 2. Apico-anterior. • Lingula (2 segments) 1. Superior. 2. Inferior.	1. Superior. 2. Antero basal. 3. Lateral basal. 4. Post. Basal.



Terms of lung diseases

1. Suppurative Lung Syndrome (SLS)

Definition

Group of diseases characterized by cough & expectoration of

- **B**ig amount (profuse).
- **P**urulent.
- **P**ostural.
- **P**aroxysmal.
- ± Foetid (**b**ad odor). بريحة

The diseases are

1. Lung abscess.
2. Bronchiectasis.
3. Infected lung cyst.
4. Empyema with bronchopleural fistula.

2. Chronic Obstructive Pulmonary Disease (COPD)

Obstructive airway diseases

Reversible (attacks)	Irreversible	
Patient in between is free, e.g. Bronchial asthma.	Asthmatic bronchitis	Chronic obstructive bronchitis
	<u>Remittent</u> : recurrent on top of wheezy chest.	<u>Progressive</u> : gradually increased.
	May be complicated by: Cor pulmonale, complication of chronic cough or respiratory failure.	

Irreversible obstructive airway diseases + emphysema = COPD

3. Cor pulmonale

Right ventricular hypertrophy and or right ventricular failure due to chest disease, on top of healthy left side.

Chest sheet

Don't forget talk about

1. History: which include
 - Personal history.
 - Complaint.
 - History of present illness.
 - Past history.
 - Family history.
 - Socioeconomically state.
 - In females (menstrual and obstetric history).
2. Examination :
 - General.
 - Local.
3. Investigations.
4. Treatment.
5. Diagnosis.

History

Personal history

1. **Name:** to be familiar with the patient. اسم حضرتك إيه ؟؟؟
2. **Age:** as certain diseases are more common in certain ages, e.g. عندك كام سنة ؟؟؟
 - T.B. more common in children and young adults.
 - Cor pulmonale and bronchogenic carcinoma more common in old age.
3. **Sex:**
 - Bronchogenic carcinoma more common in male.
4. **Occupation:** persons in certain occupations are more susceptible to certain diseases e.g.
 - **Asbestosis:** IPF – bronchogenic carcinoma or mesothelioma.
 - **Farmers:** bilharziasis – farmer's lung. بتشتغل إيه ؟؟؟
5. **Marital state:** for possible sterility or impotence.

متزوج ولا لسه ؟؟؟ عندك أولاد ؟؟؟ كام ولد ؟؟؟ أصغرهم عنده كام سنة ؟؟؟
6. **Residence:** may reflect socioeconomic condition and may occasionally point to certain disease.
 - **Helwan:** interstitial pulmonary fibrosis. ساكن فين ؟؟؟ طول عمرك ساكن هناك ولا كنت في مكان تاني قبل كده ؟؟؟
7. **Special habits:** special habit is a habit that makes the patient more susceptible than others to a certain a disease.
 - **Smoking:** chronic bronchitis, emphysema (COPD) - bronchial asthma - cancer lip and tongue - post operative pneumonia. بتدخن ؟؟؟ كام سيجارة في اليوم ؟؟؟ من إمتى ؟؟؟

Complaint

C/O + Duration

إيه اللي جابك المستشفى ؟؟؟

- By the patient own words – No medical terms.
- As short as possible (one complaint is enough).

هنشوف مثال بسيط على كده

نقول مثلاً the patient is complaining of shortening of breathing of **1 week** duration

في المثال ذكرنا إن المريض بيشكي من كُرشة نفس ،، وده من كلام المريض
ومستخدمناش مصطلحات طبية ،، وكمان كلام مختصر زي ما أنت شايف
وكمان كتبنا في الآخر ال duration ،، اللي كانت one week

History of present illness

- i. Analysis of complaint.
- ii. Symptoms of the related system.
- iii. Other systems.
- iv. Investigation & treatment (related diseases).

Characterized by:

- As long as possible
- Contains medical terms
- In chronological arrangement
- In the form of a story.

i. Analysis of the complaint**N.B.**

- For analysis any complaint in chest as usual (8) + 3 variants + 3 for any excreta.

For analysis any complaint (8) as usual

- Onset - course - duration.

onset بنسأل : الشكوى ظهرت فجأة ولا بعد أد إليه ؟؟؟

الcourse وال duration بنسأل : الأعراض ظهرت بالتدريج ولا على مدى أسابيع أو شهور ؟؟؟ أو بتحدث في نوبات

- Association.

هل كانت مصحوبة بحاجة ،، وتذكر الأعراض الثانية التي ممكن تكون موجودة ؟؟؟

- What ↑ and what ↓.

إليه التي بيزود العَرَض ده ؟؟؟ وإليه التي يقلله ؟؟

- Effect of treatment.

تأثير العلاج عليه ؟؟؟ هل لما بتأخذ العلاج الأعراض بتقل ولا بتفضل زي ما هي ؟؟؟

- Date of last attack.

آخر مرة جالك العَرَض " وتقول إسم العرض ده " كان إمتى ؟؟؟

3 variants

- Postural.

عندك بلغم بيزيد في وضع معين ؟؟ لما مَيِّل لأدام أهلاً وسهلاً إنت bronchiectasis

بيزيد لما بتنام على جنبك الشمال ؟؟؟ يبقى عنده abscess في الناحية اليمين

- Diurnal.؟؟ العَرَض ده بيزيد في النهار ولا بليل ؟؟؟
- Seasonal. ؟؟ بيزيد أو يقل صيف ولا شتاء ؟؟؟ ربيع ولا خريف ؟؟

For any excreta

وده إذا كان عنده haemoptysis or expectoration غالباً

لازم أعمل analysis لل haemoptysis وال expectoration

- Amount.
- Content - Color - Consistency.
- Odour.

ii. Symptoms of the chest

Symptoms of the chest (chest + 3 Cs + 2 Ps)

- **C**ough.
 - **H**aemoptysis.
 - **E**xpectoration.
 - **S**hortness of breathing (S.O.B.) (dyspnea).
 - **I**زيزيق (Wheeze).
 - **C**onstitutional manifestations.
 - **C**yanosis.
 - Systemic venous **C**ongestion. (Cor-pulmonale).
 - **P**ain.
 - **P**ressure manifestations.
- } CHEST
 } 3 Cs
 } 2 Ps

N.B.

- oral question in each symptom:
 - ✓ definition.
 - ✓ Analysis.
 - ✓ Causes.

1. Cough

Defined as: explosive expiration for clearing of tracheo-bronchial tree.
 “ Mainly in COPD & SLS ”

Analysis

(8) as usual

- Onset - course - duration.
- Association.
- What ↑ and what ↓.
- Effect of treatment.
- Date of last attack.

Associated symptoms:

- **Wheeze:** B.A.
- **Hoarseness:** laryngitis.
- **Vomiting:** pharyngitis.

3 variants

- Postural.
- Diurnal.
- Seasonal.
- **Nocturnal:** cardiac.
- **Early morning:** bronchiectasis & B.A.
- **All over the day:** chest infection.
- **Seasonal variation:** allergy.

+ character of cough

- **Brassy:** tracheal causes (metallic form → mediastinal syndrome).
- **Bovine:** left recurrent laryngeal nerve paralysis (hollow).
- **Barking:** hysterical.
- **Paroxysmal:** whooping cough - cavity syndrome - B.A. + pharyngeal, laryngeal and auditory irritation.

Oral questions**What are the commonest causes of chronic cough ?**

1. Chronic bronchitis.
2. S.L.S.
3. Bronchial carcinoma.
4. Pulmonary T.B. - Pneumoconiosis & sarcoidosis.
5. Post-nasal discharge as chronic sinusitis.

What are the complications of chronic cough ?

Thoracic	Extra thoracic
• Muscle (chest) pain. • Fracture rib (stress fracture). • Pneumothorax. • Emphysema. • Haemoptysis.	• Eye puffiness. • Sub-conjunctival haemorrhage. • Hernia. • Prolapse.

2. Haemoptysis**Defined** as: coughing of blood.

"Mainly in S.L.S. (50% of bronchiectasis)."

Classification

- **True:** below the vocal cord.
 - **False:** above the vocal cord.
- } Differentiated by investigations (laryngoscopy).

True haemoptysis

Type	Causes
Frothy blood tinged	A.P.O. "Acute pulmonary edema"
Blood stained	Acute infection - bronchogenic carcinoma.
Blood streaked	Chronic bronchitis - bronchogenic carcinoma - T.B.
Red current jelly (blood + sputum + hg. debris).	Bronchogenic carcinoma.
Rusty (golden brown)	Lobar pneumonia.
Frank	T.B. - pulmonary embolism - M.S. - B - bronchiectasis sicca haemorrhagica.

D.D.

	Haemoptysis	Haematemesis
Preceded by (قبل)	Cough	Nausea & vomiting
During (خلال)		
• Contents	Frothy	Food
• Color	Bright red	Dark (لون البن المحروق)
• PH	Alkaline.	Acidic
Followed by (بعد)	Blood tainted sputum	Melena.

Analysis**(8) as usual**

- Onset - course - duration.
- Association.
- What ↑ and what ↓.
- Effect of treatment.
- Date of last attack.

*History of blood transfusion,
indicates severe haemoptysis.*

3 variants

- Postural.
- Diurnal.
- Seasonal.

For any excreta

- Amount.
- Content - Color- consistency.
- Odour.

يُسأل قبله سؤال ،، وبعده سؤال

Preceded by & followed by.....

What is Bronchiectosis Sicca Haemorrhagica?

Special type of bronchiectasis caused by TB characterized by frank haemoptysis.

Explained as; TB usually affects the apical part of the lung, with good drainage so, presented with Dry cough and Frank haemoptysis.

N.B.

- *T.B. is the most serious cause of haemoptysis as it may be followed by fatal haemoptysis.*

What are the main causes of haemoptysis?

1. M.S.& L.V.F.
2. Pulmonary T.B.
3. Acute bronchitis.
4. Bronchiectasis.
5. Bronchial adenoma & carcinoma.
6. Pulmonary embolism.

3. Expectoration

Defined as: excessive spitting.

"Mainly in COPD - SLS"

Analysis

(8) as usual

- Onset - course - duration.
- Association.
- What ↑ and what ↓.
- Effect of treatment.
- Date of last attack.

3 variants

- Postural.
- Diurnal.
- Seasonal.

Postural variation: with S.L.S.

Unilateral: increased on healthy side in lung abscess.

Bilateral: increased with leaning forward in bronchiectasis.

For any excreta

- Amount.
- Content - Color- consistency.
- Odour.

> 250 cc = 1 cup.

Due to anaerobic or fungal infection

N.B.

- Postural variation can diagnose hardly the anatomical site of the lesion.

Expectoration in COPD and S.L.S.

	COPD	S.L.S.
Type	Mucoid or muco-purulent	Purulent.
Postural	No	Positive.
Association	Dyspnea - wheeze	Toxemia.

N.B. anti biotic is indicated in COPD if sputum become yellowish in color.

What are the different type of expectoration (sputum)?

1	Frothy (serous)	• Pulmonary oedema (pink frothy sputum). • Pulmonary venous congestion. • Broncho alveolar carcinoma.
2	Mucoid	• Chronic bronchitis. • Bronchial asthma.
3	Purulent Mucopurulent	• abscess and bronchiectasis. • any form of broncho pulmonary infection.
4	Rusty Golden brown	• Peumonia. (altered blood pigment)
5	Chocolate	• Amebic lung abscess (Anchovy Sauce).
6	Red-current jelly	• Bronchial carcinoma.
7	Caseous	• TB == nummular sputum (coin like).
8	Black	• Inhalation of carbon.

N.B. Excessive eosinophils can cause sputum to appear purulent like (yellow).

4. Dyspnea

May be **Obstructive (COPD)** or **Restrictive** (fibrosis).

Analysis

(8) as usual

- Onset - course - duration.
- Association.
- What ↑ and what ↓.
- Effect of treatment.
- Date of last attack.

3 variants

- Postural.
- Diurnal.
- Seasonal.

+ in C.V.S.

What are causes of paroxysmal dyspnea?

- **A** = **A**sthma (bronchial / cardiac / uraemic)
- **H** = **H**ysterical.
- **L** = **L**aryngismus stridulus.
- **A** = **A**llergic alveolitis.
- **M** = **M**yesthenia gravis / mediastinal syndrome.

What are causes of exertional dyspnea ?

- **Hypoventilation** :obstruction (COPD) or restriction (fibrosis).
- **Impairment of diffusion**: fibrosis - congestion (left sided failure).

What are causes of acute dyspnea?

1. **Hypoventilation**: obstruction (br. asthma) or restriction (pneumothorax).
2. **Impairment of diffusion**: cardiac asthma (APO).
3. **Hypoperfusion**: pulmonary embolism.

N.B. cardiac dyspnea usually exertional associated with cardiac symptoms (PND or orthopnea).

5. Wheeze

Analysis: as usual + 3 variants.

Wheeze

Continuous wheeze	Interrupted wheeze
Asthmatic bronchitis (irreversible)	Bronchial asthma

6. Chest pain

Analysis : as any pain (11 points with stress on the 3 variants)

Remember in pain there is 11 points which are

8 as usual

- Onset - Course - Duration.
- Association.
- What ↑ and what ↓.
- Effect of treatment.
- Date of last attack.

+ 3

- Site.
- Radiation.
- Character

What are the most important causes of chest pain of pulmonary origin ?

Non central	Retro sternal
• Pneumonia. • Pleural pain. • Pulmonary infarction. • T.B. • Malignancy. • Rib fracture. • Muscle strain. • Invasion of chest wall by a tumor. • Spinal nerve root involvement by vertebral disease or herpes zoster.	• Acute mediastinitis. • Mediastinal tumors. • Tracheitis. • Mediastinal emphysema.

Specific for pleural diseases:

- Characterized by: *Stitching* نَغْرٌ
- Increased with breathing, cough and straining.
- Relieved by lying on the diseased side.
- Localized (anatomical diagnosis).

7. Constitutional manifestations (toxic)

- Anorexia.
- Loss of weight.
- Fever.
- Sweating.



in S.L.S. - T.B.

8. Pressure manifestations

Mass in the chest (mediastinal syndrome).

see C.V.S.

9. Systemic venous congestion

Stress on jaundice and lower limb edema.

See C.V.S.

What are causes of jaundice in chest case?

1. **Hepatocellular**: in core-pulmonale (liver congestion).
2. **Haemolytic**: in pulmonary infarction.
3. **Obstructive**: in bronchial carcinoma (liver metastasis).

What are causes of LL oedema in chest case?

1. **R.V.F.**: core pulmonale.
2. **Hypo-albuminemia**: Excessive expectoration for a long time, or Frequent aspiration of empyema.
3. **Nephrotic syndrome**: due to renal amyloidosis in chronic S.L.S.
4. **Acid-base disturbance** in COPD (increased CO₂).

iii. Other systems

متنساخ الخطوة الثالثة Other systems
 متنسأل بقا سؤالين في ال cardio على ال Pulmonary congestion وال systemic congestion
 وال manifestation بتاعت ال low cardiac output وال syncope
 الحاجات الكبيرة دي بتسألها على السريع
 بالنسبة لل neurology بتسأل عن الحاجات الكبيرة
 motor weakness, sensory affection يعني مش بالتفصيل الممل
 ولو مفيش فيها أي حاجة
 no symptoms suggest other systems affection
 وتكتبها كده يبقا انت سألت عليها
 عشان محدش يجي يقولك أنت مسألتش على ال Other systems ليه !!!!

iv. Investigation & treatment (related diseases)

الي إنتوا بتضيعوا فيها بدون مبالغة عشر دقائق
 وهي في الآخر تسوى إيه ؟؟؟ تسوى ولا حاجة
 لو نستها ممكن تعدي
 إحنا مش هننساها إن شاء الله ومش هنضيع فيها وقت
 متقعدش تقولي العيان راح فين وجه منين ؟؟؟
 تقولي :
 Patient sought medical advice
 admitted فين ؟؟؟
 و recommended إيه ؟؟؟
 وعملوا إيه ؟؟؟
 وخرج من المستشفى
 واتحجز في المستشفى الفلاني
 وبعدين اتحجز في الثاني وبعدين مش عارف
 إحنا ملنا بالكلام ده !!!!!!!
 أنا عايز جملة واحدة
 patient investigated by ECG, chest X-ray, ECHO cardiography
 والدوبلر
 and treated by Beta blocker
 خلاص أنا مش عايز حاجة ثانية
 أنا عايزها كده جملة في الآخر
 متقعدش تفتح في حكاوي العيان ، لازم تلجم كده العيان في الامتحان
 وإلا مش هنخلص يعني ماشي ☺

SUMMARY OF HISTORY OF PRESENT ILLNESS IN CHEST

Cough

- فيه كحة؟؟؟ ظهرت فجأة؟؟ ولا إزاي؟؟؟
- كانت مصحوبة بحاجة؟؟؟؟
- إيه اللي بيرودها؟؟؟ وإيه اللي بيقللها؟؟؟
- بتزيد في وقت معين؟؟؟ بتزيد بليل ولا الصبح ولا ثابتة؟؟؟

Haemoptysis

- بتكح دم؟؟؟
- من إمتى؟؟ وظهرت فجأة ولا بالتدريج؟؟؟
- كانت مصحوبة بحاجة؟؟؟
- الكمية أد إيه؟؟ لونه إيه؟؟ الرائحة؟؟؟

Expectoration

- عندك بلغم زيادة؟؟؟
- بيزيد في وضع معين لما يتميل لأدام مثلاً؟؟؟ ولا بيزيد لما بتنام على جنبك؟؟؟
- بيزيد في موسم معين أكثر من الثاني؟؟ بيزيد في الصيف عن الشتاء؟؟؟
- الكمية أد إيه؟؟؟ لونه إيه البلغم؟؟ والرائحة؟؟؟

Dyspnea (Shortening of breathing)

- عندك كرشية نفس؟؟؟

Wheeze (تزييق)

- عندك تزييق في صدرك؟؟؟

Chest pain

- عندك ألم في صدرك؟؟؟

Pressure manifestations

- هل صوتك اتنبح؟؟؟
- هل عندك صعوبة في البلع؟؟؟

Constitutional symptoms

- عندك فقدان للشهية؟؟؟ وزنك خَس؟؟؟ بتعرق كثير؟؟؟

Systemic venous congestion

- رجلك أو بطنك تَفَخَت؟؟؟
- فيه ألم في جنبك اليمين؟؟؟

Cyanosis

- إزرقيت؟؟؟ حصل زُرقة؟؟؟

وتسأل المريض برديو

وتقوله :

هل عندك مشاكل في أي جزء ثاني في جسمك؟؟؟

عملت فحوصات إيه؟؟

وأخذت علاج إيه؟؟؟

Past history

1. T.B.
2. D.M. (T.B. follows D.M. as its shadow).
3. Similar attack.
4. Asthma.
5. Bilharziasis.
6. Chest trauma.
7. History of anesthesia or coma (aspiration).

Family history

1. History of chest infection e.g. T.B.
2. Similar conditions.
3. History of allergy (asthma).
4. History of D.M. (T.B. follows D.M. as its shadow).

Examination

General

A أرقام	• Hyper tension: poly arteritis nodosa (associated bronchial asthma). • Hypotension: miliary T.B. (causing Addison's disease).
B	• Built ✓ Under built: chest disease since birth (T.B.). ✓ Over built: Pichwichian syndrome.
C	• Color ✓ Cyanosis: respiratory failure. ✓ Jaundice: liver affection.
D	• Decubitus ✓ Lateral position: in unilateral chest disease (e.g. Trepopnea).
E	• Neck vein – H & N (total) حاجة فوق ✓ Congested neck vein due to : ☞ Cor-pulmonale. ☞ Massive pleural effusion or pneumothorax. ☞ Chronic obstructive pulmonary disease. ☞ Mediastinal syndrome.
	• Clubbing (upper limb) حاجة في النص ✓ Chronic suppurative lung disease. ✓ Bronchogenic carcinoma. ✓ Chronic obstructive pulmonary disease. ✓ Interstitial lung fibrosis. ✓ Mesothelioma. • Flapping tremors in respiratory failure (CO₂ retention).
F	• Lower limb edema (lower limb) حاجة تحت
	• Mentality "فكر" ✓ Disturbed mentality (CO₂ narcosis). • Face + general look. ✓ Puffiness (chronic cough).

Enlarged lymph nodes due to:

- Bronchogenic carcinoma (Scalene lymph nodes).
- Tuberculosis.
- Sarcoidosis.

Other systems examination**C.V.S. examination**

Cause	Result	Association
- Pulmonary congestion. - Pleural effusion. - Left sided failure (BBC).	Cor-pulmonale.	Kartagner's syndrome.

Abdomen examination

Liver (hepatomegaly)	Spleen (splenomegaly)	Ascites
- Cor-pulmonale. - Hypoxic cor-pulmonale. - Ptosed liver (emphysema). - Amoebic liver abscess. 2ry from Br. Carcinoma. - Fatty changes (toxemia). - Association.	- Cor-pulmonale. - Hypoxic cor-pulmonale. - Miliary T.B. - Sarcoidosis. - Amyloidosis. - Associated.	- Cor-pulmonale. - T.B. peritonitis.

Ascites:

- Ascites + Rt pleural effusion = suggestive to liver cirrhosis.
- Ascites + left pleural effusion = suggestive to lung disease e.g. T.B.

Local examination

Introduction: lines and areas of the chest.

Lines of the chest

Front	Lateral	Back
Mid line: middle of the sternum	AAL: anterior fold of the axilla.	Midline: spines of the vertebrae.
Parasternal: midway between MCL and lateral border of the sternum.	MAL: apex of the axilla.	Paravertebral: midway between midline and medial aspect of the scapula.
MCL: midway between the acromion process and sternoclavicular joint (as Mid way of inguinal point).	PAL: posterior fold of the axilla.	Scapular line: from the angle of the scapula.

Areas of the chest

Front (3)	Lateral (2)	Back (3)
• Supra-mammary. • Mammary. • Infra mammary.	• Upper lateral. • Lower lateral.	• Supra scapular. • Inter scapular. • Infra scapular.

N.B.

- Examination of the back in chest cases is important
- **Inspection & palpation:** Area with area.
- **Percussion:** space by space
- **Auscultation:** Area with area or space with space.

Local examination

	Inspection (6)	Palpation (5) (T)	Percussion (4)	Auscultation (3)
S	1. Shape of the chest.	1. <u>T</u> rachea.	1. Lung proper.	1. Breath sound.
M	2. Respiratory movement.	2. <u>T</u> enderness.	2. Bare area.	2. Addventious sounds.
S	3. Skin lesion.	3. <u>I</u> .V.F.	3. Traub's area.	3. Vocal Resonance.
T.	4. Trill's sign.	4. Palpable wheeze. تزييق	4. Kronig's isthmus.	
P.	5. Pulsations.	5. Expansion تمدد		
±	6. Littin's sign.			

i. Inspection (SMS T.P.)

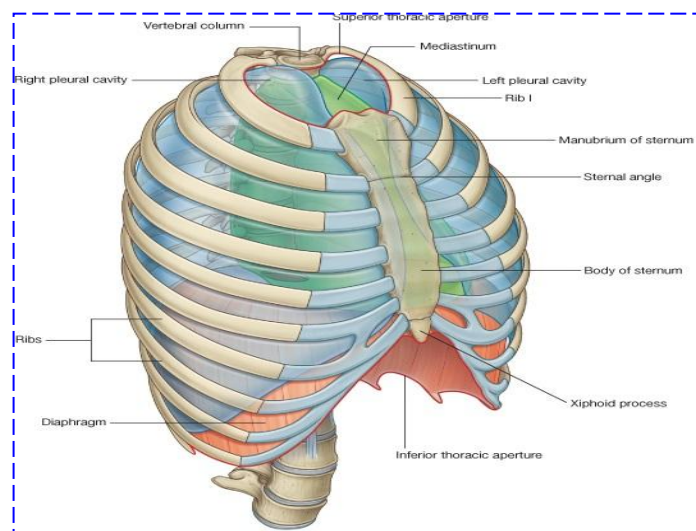
النظر من عند قدم المريض مماسياً، « وقل للمريض : خذ نفس

1. **Shape of the chest**

- Diameters (normal = antero posterior : transverse = 5 : 7).
- Symmetrical or not.
- Thoracic cage.

thoracic cage مفردات ال

- sub-costal angle ← تحت ♦
- shoulder ← فوق ♦
- back ← خلف ♦
- sternum ← أمام ♦
- Ribs ← يمين وشمال ♦



Abnormal shapes

A. **Barrel chest**: "Emphysematous" as in **COPD**

- ❖ Antero posterior $\geq$ transverse.
- ❖ Ribs: transverse with wide I.C. spaces.
- ❖ Thick shoulder.
- ❖ Sub-costal angle: wide.
- ❖ Protrusion of the sternum.
- ❖ Kyphosis.



B. **Funnel chest**: "Pectus excavatum"
Congenital or occupational.

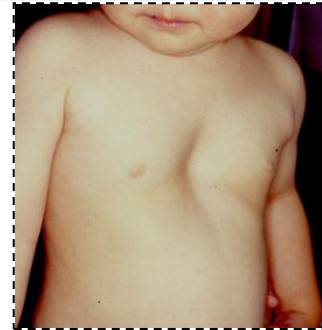
C. **Pigeon chest**: "Pectus carniatum"
In Rickets.

D. **Unilateral retraction**:
Fibrosis & collapse.

E. **Unilateral bulge**:
 ➤ Pleura → effusion, pneumo-thorax.
 ➤ Chest wall → emphysema, tumors.

N.B.

- To differentiate between retraction and bulging look at the moving side which is the healthy one.



2. Respiratory movement

- **Rate**: (12 - 20 cycles per min.) (pulse / Res. = 4 / 1).
- **Rhythm**: regular.
- **Depth**: average, deep or shallow.
- **Type**: normally; abdomino thoracic (male) - thoraco abdominal (female).
- **Degree of chest expansion**: limited in all chest diseases.

3. Chest wall (Skin) lesion

Scars, dilated veins & pigmentationsetc.

4. Position of the trachea (mediastinum):

Prominent sternomastoid tendon on the shifted side (Trill's sign).

5. Pulsations:

- Apex: absent in emphysema.
- Epigastric & left parasternal pulsations (RV ++).

6. Littin's:

The diaphragmatic shadow on chest wall.

- Normally: in thin patients.
- Its presence exclude:
 - ❖ Paralysis of diaphragm.
 - ❖ Pleural effusion.

N.B.

Hoover's sign: inward movement of the ribs in advanced COPD.

ii. Palpation (5 Ts)

1. Trachea:

Trachea is clinically central, but radiologically shifted to the right because of aortic arch.

Technique:

- Put the patient in the sitting position.
- Fix his head by your left hand.
- Insert your index in the pouch between the two sternal ends of sternomastoid.

What are causes of tracheal shift?

Tracheal shift

To the same side of lesion	To the opposite side of lesion
Fibrosis, collapse or pneumonectomy.	Pleural effusion, pneumothorax, hydropneumothorax or unilateral emphysema.

Add to tracheal examination

Crico-sternal distance: distance from the cricoid to the suprasternal notch = 4 - 5 cm. this length may be shorten in COPD. (insert one or two fingers between the cricoid cartilage and the suprasternal notch).

Tracheal tug

A) Campbell's sign = tracheal tug with inspiration: Trachea descent with inspiration in severe COPD (It is not specific).

Explained by pulling on the trachea during inspiration due to strong diaphragmatic contraction in COPD.

B) Oliver's sign = tracheal tug with pulsation: descent of cricoid cartilage with ventricular contraction in cases of aortic aneurysm.

2. Tenderness: For any chest lesion.

Tenderness according the site

Over sternum	Over spaces	Over ribs	Right side
Leukemia.	Emphysema.	Fracture or osteomyelitis.	Amoebic liver abscess.

Examine front, lateral and back: area with area.

3. T.V.F.

Definition: vibration of the vocal cords transmitted through airway & chest wall to be palpated on the chest wall.

Technique:

- Ask the patient to say 99 (44 in arabic)

لو المريض أجنبى ، خليه يقول : nine, nine (ninety nine)
لو المريض عربى ، خليه يقول : أربعة وأربعين ، أو أربعة أربعة

- Palpate the chest: area with area.

Causes of increased T.V.S.: 3 Cs

- **C**onsolidation.
- **C**ollapse with patent bronchus.
- **C**avity if superficial & surrounded by consolidation.

N.B.

- T.V.F. is reduced in any other chest diseases.

Physiologically higher in the 2nd right space, why???

As the right bronchus is more superficial.

4. Palpable wheeze تزييق**Technique:**

- With deep respiration.

Causes in COPD.

D.D.: with thrill by stopping breathing.

5. Chest expansion تمدد**Technique:**

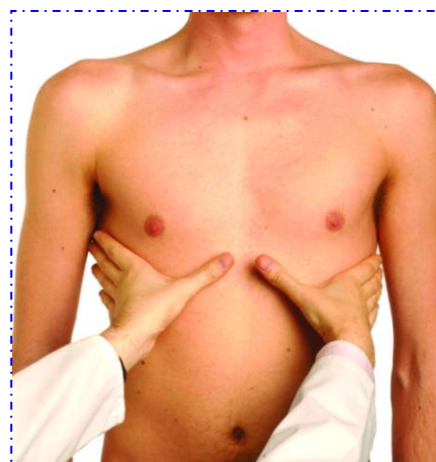
- Front: all areas.
- Back: supra-scapular & infra scapular areas.

Causes of limitation of chest expansion:

- Bilateral: COPD.
- Unilateral: fibrosis & collapse.
- Bilateral: bronchoiectasis.

N.B.

- Chest expands about 5 - 7 cm تقاس بالمازورة

**iii. Percussion****Principle and technique**

air is resonant, everything else is dull

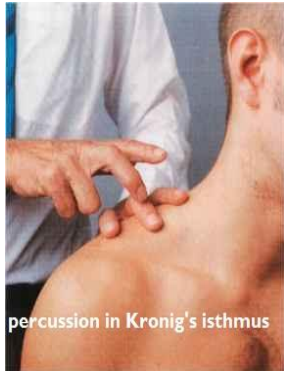
- We use the middle finger of the right hand, it struck sharply and quickly with the tip over the shaft of the middle phalanx of the left middle finger.
- Place the left hand on the chest wall with the fingers parallel to the ribs, the middle finger is placed firmly in the inter-costal space chosen.
- Light percussion gives palpable rather than audible vibration.
- Heavy percussion gives palpable and audible vibration.



Types of percussion notes:

1. **Tympanic:** hollow viscous e.g. normal traub's area.
2. **Hyper-resonance:** emphysema and pneumothorax.
3. **Resonance:** normal lung.
4. **Impaired note:** pulmonary consolidation or fibrosis.
5. **Dullness:** pulmonary consolidation, pulmonary collapse & fibrosis.
6. **Stony:** pleural effusion.

Percussion of the lung

A. Lung proper			B. Special area
Front	Lateral	Back	1. Bare area. 2. Traub's area. 3. Kronig's area.
1. Calvicular (direct). 2. Infra clavicular. 3. Space with space at MCL except left 4 th & 5 th spaces at AAL. 4. Ends at the Rt. Side by hepatic dullness (tidal P.) ends at the Lt. side by tympanitic resonance of traub's area.	At MAL space by space.	Started by Kronig's isthmus. Then, 1. Supra scapular. 2. Inter scapular. 3. Infra scapular. Alternative, Space by space	

Special areas:**1. Bare area:**

Definition: area of the heart not covered by the lung.

Anatomy: left 4th & 5th inter-costal spaces, from the left border of the sternum to the left parasternal line.

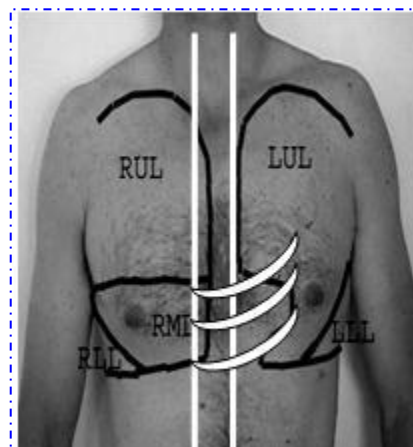
Normally: dullness (blood).

Abnormalities:

- Resonant:
 - Emphysema (the surest sign).
 - Pneumothorax.
- ↑ dullness of the bare area in:
 - Collapse.
 - Rt. V ++.
 - Pericardial effusion.

Technique: (light percussion)

- Classic: from outwards inwards.
- Alternative from above downwards.



2. Traub's area

Definition: area of the chest overlying the air bubbles at the fundus of the stomach.

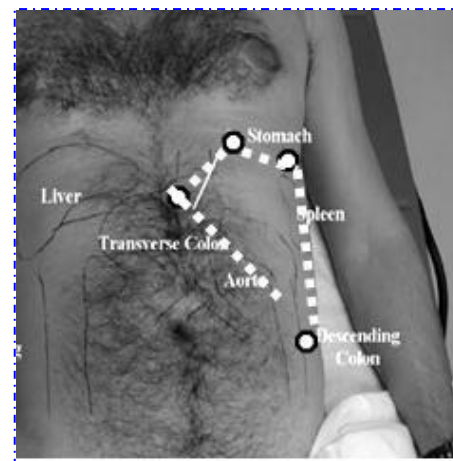
Anatomy:

1. 5th rib at MCL.
2. 9th rib at MAL.
3. Costal margin at MAL.
4. 8th costochondral junction.

Normally: tympanatic resonant. (air bubbles).

Abnormalities:

- Dullness:
 - Above: pleural effusion & pericardial effusion.
 - Down: ascites, pregnancy and tumors.
 - Right: hepatomegaly.
 - Left: splenomegaly.
 - Full stomach or gastric tumor.
- Wide area:
 - Lobectomy, splenoectomy.
 - Shrunken liver or dilated stomach.



Technique:

1. From above downwards at AAL. Or,
2. From out inward.

3. Kronig's isthmus

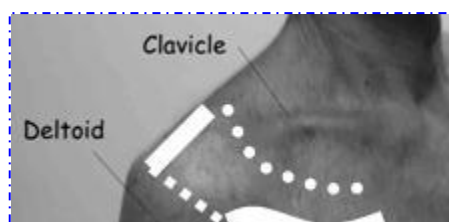
Definition: a band of resonance overlying lung apex.

Anatomy:

1. Anterior: clavicle (medial 2/3).
2. Posterior: trapezius muscle (medial ½).
3. Medial: root of the neck.

Bony border:

- Anterior: medial 2/3 of the clavicle.
- Posterior: medial ⅓ of the spine of the scapula.
- Lateral: a line connects 2 points:
 1. Junction between medial 2/3 & lateral ⅓ of the clavicle.
 2. Junction between medial ⅓ & lateral 2/3 of the spine of the scapula.
- Medial: a line connects sternoclavicular joint and C₇.



Normally: resonant.

Abnormalities: dullness in apical lesions as:

1. T.B.
2. Pancost tumor.
3. Friedlander's pneumonia.

4. Fibrosis / collapse.
5. Pleural thickness.

Technique: from the back while the patient in sitting position.

- From in outward.

Values of Tidal Percussion

1. **Differentiate** supra from infra diaphragmatic dullness (see the lower table)
2. **Diaphragmatic movement:** measuring the distance between the lower border of pulmonary resonance in full inspiration and forced expiration at the back of the chest.

Normal: dullness at T10 changed to resonance by inspiration

Abnormalities

Infra diaphragmatic	Reversed tidal percussion	Supra diaphragmatic
Dullness above T10 changed to resonance by inspiration.	Resonant becomes dull on inspiration (diaphragmatic paralysis).	Dullness above T10 (didn't change).

Shifting dullness

Used to differentiate pleural effusion, from hydro pneumothorax by percussing in 3 planes.

- *In pleural effusion no change while in hydro pneumothorax it is changed.*

1st plane: Percuss the M.C.L from above downward till the dullness, proceeds one space above (resonant), ask the patient to sit, the resonance will be dull in hydro pneumothorax).

2nd plane: Percuss from the sternum to the axillary line till the dullness, ask the patient to lie on the opposite side. The dullness will be resonant in hydro pneumothorax.

3rd plane: Percuss the back (scapular line) from above downward till the dullness, ask the patient to lean forward. The dullness will be resonant.

Physiological introduction

A. Breath sounds are normally produced by 2 elements:

1. Laryngeal element: vibrations of the vocal cords "glottis hiss".
2. Vesicular or lung element: vibrations of the peripheral lung tissue by inspiration only.
 - **Inspiration** = glottis hiss + the periphery.
 - **Expiration** = the glottis hiss only.

N.B.

- Bronchial breathing is glottic due to transmission of the glottic hiss through bronchial tree to an area with alveoli out of function.
- The normal alveoli modify the bronchial sound into vesicular.

B. Muscles of respiration:

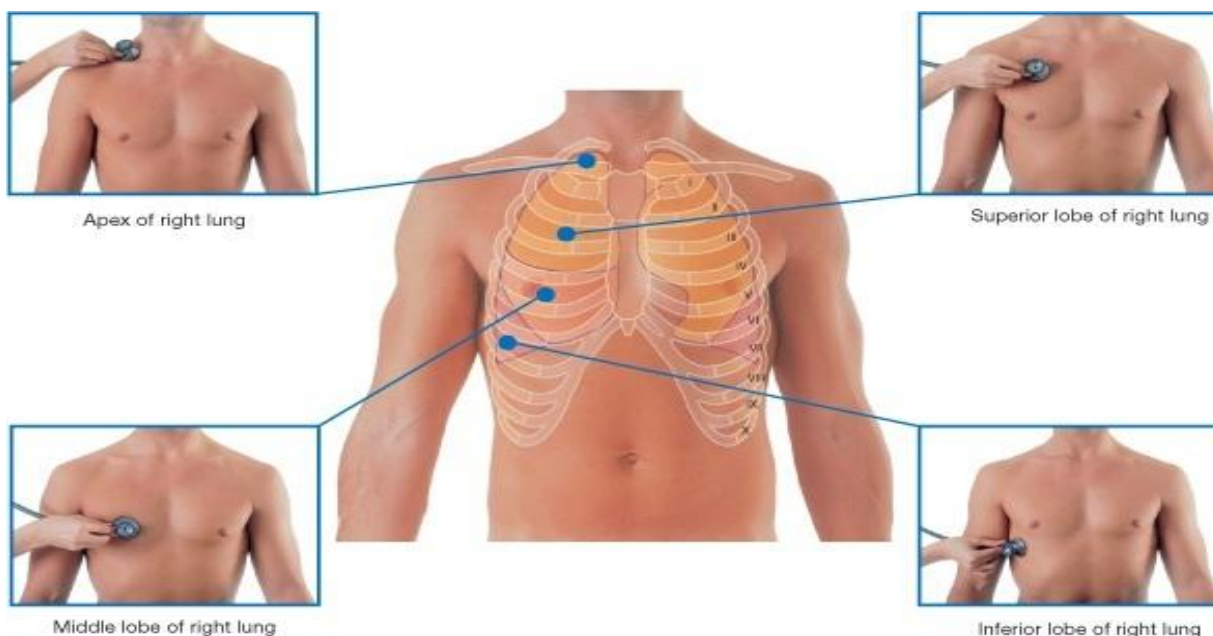
Normal	Accessory	
• Diaphragm. • Inter-costal muscles. "only for inspiration as expiration is a passive process".	Inspiratory muscles	Expiratory
	• Sternomastoid. • Trapezius. • Scalnii.	• Abdominal muscle. • Latesmus doraslis. • Lips.

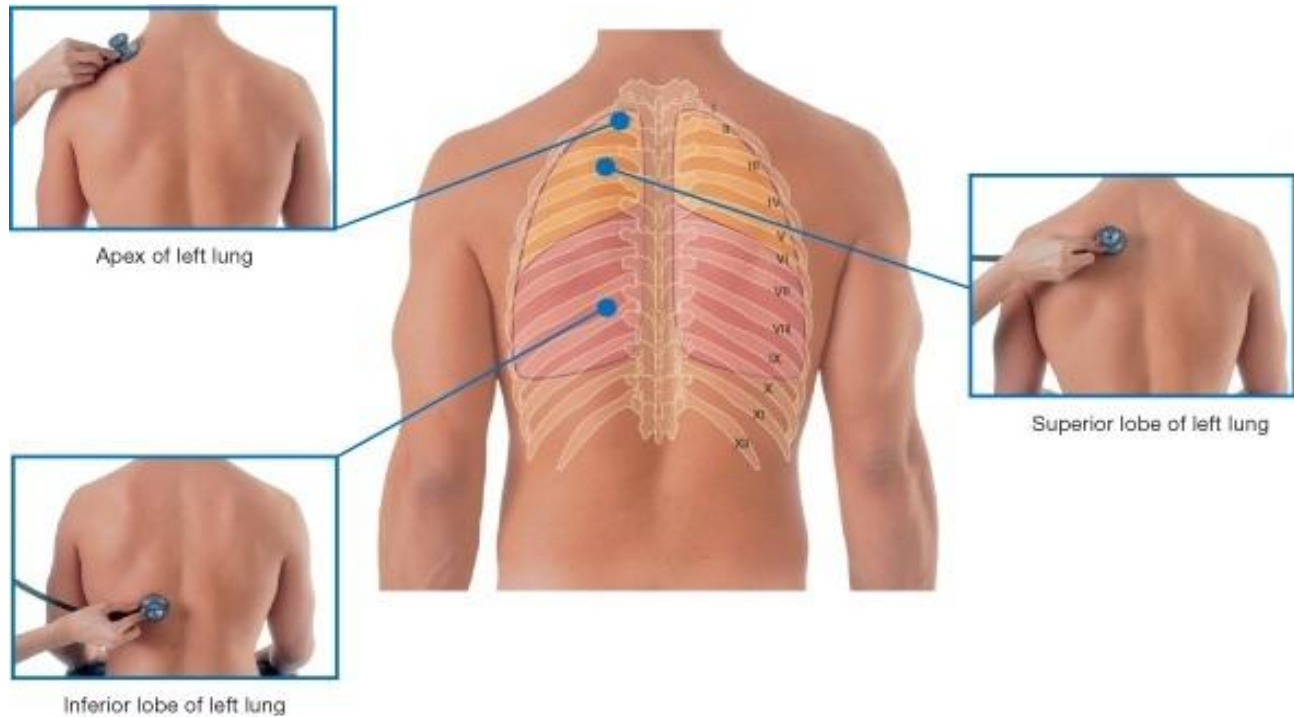
N.B.

- Usage of accessory muscle means difficult breathing in cases of airway obstruction as COPD.

iv. Auscultation

- A. Air entry.
- B. Breath sounds.
- C. Additional sound.
- D. $\pm$ vocal resonance.





- We can use either diaphragm or bell (cone).
- Most of the sounds reaching the chest wall from the bronchi and lungs are of low frequency so, the bell is preferred.
- Stretching of the skin and hairs under the diaphragm during deep breathing produce sounds like pleural rub or crepitations so the bell is preferred.
- The patient is relaxed and breathing deeply and fairly rapidly through the mouth.
- Avoid prolonged deep breathing to avoid giddiness or tetany.

A. Air entry

Causes of decrease air entry

Reduced air flow	Reduced conduction
• Severe C.O.P.D. • Collapse.	• Pleural disease. • Thick chest wall.

B. Breath sounds

	Vesicular breathing (V.B.) (alveolar breathing) The alveoli are functioning.	Bronchial breathing (B.B.) The alveoli are out of function
Character	• Soft. • No gap. • Inspiration > Exp.	• Hollow. • Gap. • Insp. ≤ Exp.
Causes	A. In normal persons. Vesicular breathing with prolonged expiration (harsh vesicular): • Obstructive airway as; COPD. N.B. but there is may be diminished breath sound in severe COPD.	A. Normally on trachea. B. Abnormally in (3 Cs): • Consolidations. • Collapse with patent bronchus. • Cavity if superficial & surrounded by consolidations. Subtypes of B.B. are tubular, cavernous & amphorous.
		

N.B. subtypes of Bronchial breathing are: tubular, cavernous & amphorous

Tubular	Cavernous	Amphoric
High pitched sound	Low pitched sound	Low pitched sound
• Consolidations. • Collapse with patent bronchus. • Cavity if superficial & surrounded by consolidation.	Cavity: • T.B. • Abscess.	Cavity with tense wall • Tension pneumothorax. • Large superficial cavity. • Bronchopleural fistula.

N.B.

- **Despin's sign:** bronchial breathing below the level of trachea "4th thoracic vertebra" due to enlargement of the inter bronchial lymph node.

Causes:

- T.B.
- Carcinoma.

C. Additional sounds

1. Rhonchi.
2. Crepitations.
3. Rub.

In any additional sounds:

- Distribution.
- Timing.
- Type.
- Effect of cough.

1. Wheeze (Rhonchi)

Definition: it is continuous musical sound.

Causes: bronchi or bronchial obstruction by:

- Spasm of the wall.
- Mucous in the lumen.
- Pressure from outside by L.N. enlargement.

So, it occurs in B.A. & C.O.P.D. due to spasm & mucous secretion.

Character of rhonchi in COPD:

- Generalized (bilateral).
- Inspiratory & expiratory but mainly expiratory.
- Changed with cough.
- Polyphonic.

❖ **Siblent:** distal small bronchioles "**high pitched**" e.g. bronchial asthma.

❖ **Sonorous:** central small bronchioles or bronchi "**low pitched**" e.g. chronic bronchitis.

N.B.

- Wheezes may be heard in fibrosis.
- Localized, monophonic, fixed wheeze is specific for tumor.

2. Crepitations "Rales"

Definition: it is an interrupted moist non musical sound.

Mechanism of crepitations:

1. Air passage "Bubbles through fluid".
2. Reduction of the lung elasticity.
3. Fluid in the interstitium.

N.B.

- Early inspiratory crepitations may occur in COPD, called **opening snap of the chest**, but late inspiratory crepitations indicate infection.

Causes and types

Fine "8 cycles / min"	Medium "3 - 7 cycles / min"	Coarse "1 - 2 cycles / min"
• Early pneumonia. • Early T.B. • Early interstitial lung disease.	• Pneumonia. • T.B. • Lung abscess. • Bronchiectasis.	• Late pneumonia. • Late T.B. • Lung abscess. • Bronchiectasis.
• BBC		• APO

Consenting or non consonatins.

Consenting = metallic tone "in medium sized crepitations = surrounded by consolidations".

3. Rub "pleural rub"

Definition: it is a stitching friction gritty sound, disappears by holding of breath.

Causes:

- Pleurisy (dry or with effusion).

D.D.:

- Pericardial rub (with heart beats).
- Friction of the stethoscope: disappears by firm pressure.

D. Vocal resonance

Definition: it is vibrations of the vocal cord transmitted through airway and chest wall to be auscultated.

Called: **voice sound**

- Diminished in all chest lesions except: 3 Cs
 - Consolidations.
 - Collapse with patent bronchus.
 - Cavity if superficial & surrounded by consolidations.

Method: patient say 99 " 44 in Arabic"

1. **Bronchophony:** in a loud voice.
2. **Whispering pectrology:** by whispering "diagnostic".

N.B.

- **Aegophony** is heard in upper border of pleural effusion with nasal tone.
- **E to A changes:** is a variant of aegophony.

متنشاش 😊 !!!!!

N.B.

- Examination of the back in chest cases is important
- **Inspection & palpation:** Area with area.
- **Percussion:** space by space
- **Auscultation:** Area with area or space with space.

Diagnosis

1. Aetiological.
2. Anatomical
3. Pathological .
4. Functional.

1. Aetiological

Irritation	Allergy	Infection
By personal history: - Smoking. - Occupational H. - Residence.	Attacks with the same precipitating factors and the same relieving.	As T.B. - Chest troubles. - Toxic symptoms. - Multiple drugs for prolonged duration.

2. Anatomical

By history (see natural history) + local examination + investigation (see later).

Anatomical diagnosis: rarely reached in history taking as,

1. Expectoration with postural variation (SLS).
2. Site of chest pain.

3. Pathological

By natural history as following

Natural history

COPD	- Cough & expectoration. - Dyspnea. - Wheeze.
SLS	Cough & expectoration (Ps). (haemoptysis in bronchiectasis - toxic symptoms in lung abscess).
Cavity	No symptoms - only examination.
Fibrosis	Mainly post T.B. - Manifested with dyspnea.
Pleural effusion	- Pleural pain (أهمهم) - Pleurotic cough. - Dyspnea. - History of paracentesis

- Amount.
- Aspect (clear - turbid).
- Color.
- Complications (infection fistula).
- Recurrence or not.

4. Functional

A. Compensation

Respiratory failure diagnosed by

- ✓ Cyanosis.
- ✓ Flapping tremors.
- ✓ Mentality changes.

But ABG is mandatory

B. Complication

➤ **Corpulmonale** diagnosed by

- ✓ Long history of lung diseases.
- ✓ Right ventricular enlargement (5 signs).
- ✓ Right ventricular failure (L.L. edema - neck vein - enlarged tender liver).

➤ **Chest infection**

➤ **Complications of chronic cough**

فوق	Puffiness - sub-conjunctival hemorrhage.
منتصف	Chest pain - hernias.
تحت	Rectal prolapse.

Investigations

Scheme for investigations

1. Aetiological	2. Anatomical	3. Functional + complications	4. Pathological
➤ Allergy: skin test. ➤ T.B.: tuberculin test - CBC - ESR - recently PCR.	➤ Radiological e.g. Plain X-ray.	➤ Cor-pulmonale: see C.V.S. ➤ Respiratory failure: arterial blood gases.	➤ Laboratory. ➤ X-ray. ➤ C.T. - MRI. ➤ Ultra sound. ➤ Pulmonary function tests. ➤ Endoscopy. ➤ Biopsy.

1. Laboratory

A. Sputum analysis:

Indications: all cases (to detect culture & sensitivity).

- In **S.L.S.**: culture & sensitivity in necessary.
- In **COPD**: in about 90 % of cases the causative organism is well known (H.influenza or strept. Pneumoniae).

But culture & sensitivity is indicated in the following cases:

1. Failure of empirical antibiotics.
2. Associated with bronchiectasis.

B. Serous fluid analysis (pleural aspiration)

In cases of pleural effusion.

C. Sweat analysis

In cases of cystic fibrosis

D. Serum α 1 antitrypsin:

In cases of primary emphysema

2. Chest X-ray

Plain X-ray	Bronchography
See volume IV Value: for pleural effusion.	For bronchiectasis. Replaced nowadays by HRCT.

3. C.T. scan

Of choice in abscess & cavity.

4. Ultra sound

Only for encysted pleural effusion.

5. Pulmonary function tests (PFT)

For accurate diagnosis	For follow up	Bed side P.F.T.
By Spirometry 1. F.V.C. = 5 liters. 2. FEV1 = 4 liters. 3. TVC = FEV1 / FVC = 80 %.	By Flowmeter PEFR = 400 - 600 L / min. أعلى معدل لخروج هواء الزفير خلال دقيقة	Match (candle) test = 15 cm. Timed expiratory test: by stethoscopy on trachea "exp." 4 sec.

Values:

- **COPD** = obstruction pattern.
- **Fibrosis** = restrictive pattern.

6. Endoscopy (bronchoscopy)

Diagnostic	Therapeutic
1. Visualize the lesion. 2. Biopsy.	1. To remove F.B. or mucous plug. 2. Instillation of antibiotic in abscess or cytotoxic drug in malignancy.

7. Biopsy

In pleural effusion for diagnosis of pleural T.B.

N.B. types of respiratory failure:

- **Type A** = hypoxia ($O_2 < 60$ mmHg).
- **Type B** = hypoxia & hypercapnia ($O_2 < 60$ mmHg & $CO_2 > 50$)

Summary

COPD	➤ Pulmonary function tests (obstructive).
Fibrosis	➤ Pulmonary function tests (restrictive).
Bornchiectasis	➤ At past: bronchography. ➤ And now: HRCT.
Cavity & abscess	➤ At past: convent. Tomography. ➤ Now: C.T.
Pleural effusion	➤ Detection by plain X-ray. ➤ Encysted by ultra sound. ➤ T.B. need biopsy

Hypoventilation

	Obstructive e.g. Ch. Obstructive bronchitis	Restrictive e.g. fibrosis
V.C.	----	----
FVC1	----	----
TVC	----	+++
Residual volume	+++	----

Cases

Chronic obstructive Pulmonary disease (COPD)

Definition

Chronic obstructive bronchitis & emphysema commonly coexist and the condition is known, chronic obstructive pulmonary disease (COPD).

- The airflow limitation is usually progressive & not fully reversible.

Aetiology and pathophysiology

	Chronic obstructive bronchitis	Emphysema
Aetiology	1. Irritation. 2. Allergy. 3. Infection.	False: Senile, compensatory or congenital True: <u>Primary:</u> α 1 antitrypsin deficiency. <u>Secondary:</u> Infections – obstruction.
Respiratory dysfunction	➤ Obstructive hypoventilation. ➤ V/Q imbalance. ➤ Hypoxaemia & hypercapnia. ➤ Later on pulmonary hyper-tension & cor-pulmonale. ➤ Hypercapnia decreases the density of respiratory centre. This leads to absence of severe dyspnea. ➤ Type B, bronchitic Type or "Blue Bloater".	➤ Diffusion defect (decreasing surface area of alveoli). ➤ Increased V/Q . ➤ Hypoxaemia with normo or hypocapnia. ➤ Sensitivity of respiratory centre is normal (no hypercapnia) dyspnea. ➤ Type A, emphysematous Type or "Pink puffer".

Cardiovascular abnormalities

Pulmonary hypertension leads to cor-pulmonale

	Type A	Type B
Dyspnea	More marked	Less marked
Sputum	Scanty, mucoid	Copious, purulent
Weight	Decreased	Normal or increased
Cyanosis	Mild or absent	Marked
Cor pulmonale	Late	Common
Chest signs	Hyperinflation	Increased airway resistance

N.B.

- *Hypoxia: resulting in vasoconstriction of pulmonary arterioles.*
- *Reduction of the pulmonary vascular bed.*

Clinical picture**Symptoms**

Patient	Male, chronic heavy smoker & above 50 years
Bronchitis	Long history of chronic cough with mucoid or mucopurulent expectoration.
Emphysema	Dyspnea is gradual progressive occurring initially on exertion but later at rest $\pm$ wheeze.
Complications	➤ Chest pain (Chronic cough, pneumothorax). ➤ Respiratory failure. ➤ Oedema of lower limbs.

Signs**General examination (A, B, C, E & F)****Vital signs**

- Pulse
 - ✓ **Tachycardia & big pulse volume** .
hyperdynamic circulation of hypercapnia & hypoxaemia.
 - ✓ **Small pulse volume**
Severe pulmonary hypertension $\pm$ heart failure.
 - ✓ **Pulsus paradoxus**
May be present in severe cases.
- Respiratory rate
 - ✓ *Tachypnea with working accessory respiratory muscles.*

Head & neck examination

- ✓ *Central cyanosis may be present.*
- ✓ *Puffiness of eyelids due to chronic cough.*
- ✓ *Congested neck veins (increased intrathoracic pressure – Cor pulmonale & right-sided heart failure).*

Upper limb examination

- ✓ *Clubbing if there is associated bronchiectasis.*

Lower limb examination

- ✓ *Oedema of the lower limbs.*

Abdominal examination

✓ *Palpable liver due to:*

- Depression of the liver by flat diaphragm (not tender).
- Congested liver due to cor pulmonale (tender).
- Ascites may be present: due to right-sided heart failure.

Signs of respiratory failure may present

(see above)

Chest examination

Inspection	Palpation	Percussion	Auscultation
➤ Barrel chest. ➤ Bilateral limitation of expansion. ➤ Weak or absent cardiac pulsation.	➤ Trachea is central. ➤ T.V.F. is decreased bilaterally.	➤ Hyper-resonance. ➤ Encroachment on cardiac & hepatic dullness.	➤ Vesicular breath sounds with prolonged expiration. ➤ Generalized wheezes (rhonchi). ➤ Minimal crepitations may occur
Hyper inflation			Air way obstruction

Complications

Local	Systemic
➤ Respiratory failure. ➤ Pulmonary infections. ➤ Bronchial obstruction. ➤ Pneumothorax. ➤ Complications of chronic cough.	➤ Cor pulmonale. ➤ Erythrocytosis. ➤ Thrombembolism. ➤ Left-sided heart failure. ➤ Salt & fluid retention. ➤ Proteinuria. ➤ Peptic ulcer.

Investigations**Chest X-ray**

- Hyper inflation.
 - Hypertranslucency of the lungs.
 - Transverse & wide intercostals spaces.
 - Low flat diaphragm.
 - Ribbon-shaped heart.

Exaggerated broncho-vascular markings (Ch. Obstructive bronchitis)

Respiratory function tests

➤ Ventilation tests:

- Residual volume (RV) is increased.
- Forced vital capacity (FVC) usually decrease.
- FEV1, timed vital capacity & PEFr are decreased.

Diffusion tests: CO transfer factor is decreased

Arterial blood gases

Decreased PO₂ - increased PCO₂

Sputum culture & sensitivity

May detect organisms especially pneumococci & H. influenza.

Blood picture

May show erythrocytosis.

Treatment

1. Prevention.
2. Symptomatic treatment.
3. Treatment of complications.
4. Oxygen therapy.
5. Lung transplantation.

1. Prevention

- Stop smoking: is vital even in late stage on the disease.
- H. influenza and pneumococcal vaccination.

2. Symptomatic treatment

- Mucolytics e.g. bromhexine hydrochloride (bisolvon).
- Expectorants e.g. potassium iodide.
- Bronchodilators :
 - ☞ Short acting:
 - Short acting Beta 2 agonist e.g. salbutamol.
 - Short acting anti-cholinergic agents e.g. ipratropium bromide.
 - ☞ Long acting:
 - Long acting Beta 2 agonist e.g. salmeterol: given twice daily by inhalation.
 - Long acting anti-cholinergic agents (tiotropium bromide): once daily.

- Thiophylline.
- Corticosteroids. (in acute exacerbation or in patients with overlap asthma).
- Antibiotics.
- Alpha one antitrypsin replacement (congenital emphysema).

3. Treatment of complications

- Respiratory failure.
- Heart failure.
- Erythrocytosis.

4. Oxygen therapy

- It is indicated in:
 - ☞ Severe hypoxia. $PO_2 < 40$ mmHg with exertion. Or < 55 mmHg at rest.
- Value:
 - ☞ Decrease complications.
 - ☞ Improve symptoms, exercise tolerance, quality of life & survival.

5. Lung transplantation

Is the only radical treatment.

الموضوع برؤية أخرى ☺

COPD

chronic obstructive lung disease معناها إنه فيه
اللي هو شامل

• ال chronic bronchitis

• وال emphysema في أن واحد

بس لازم فيه Character رئيسية ،، اللي هي decreased in forced expiratory one
اللي هو معدل خروج ال expired air في أول ثانية في ال expiration

ال chronic bronchitis دي ،،

بتكون مميزة بإنها chronic productive cough لمدة لا تقل عن سنتين متتاليتين ،، ولمدة حوالي تلت أشهر في السنة
بس لازم تعمل exclusion لمرض مشابه جداً اللي هو ال bronchiectasis

ال **etiology** بيكون عبارة عن chronic irritation وليس chronic infection ،، وده عن طريق ال smoking وال air
،، pollution

بس أهم حاجة ال smoking

وخلي بالك ،، ال smoking مش مجرد إنها irritant بس ،، دي هي trigger the release of cytokines
يعني هتلاقى ال Interlukin وال tumor necrosis factor والحاجات دي بتبقا released مع ال chronic smoking
غير إن التدخين بيبوظ ال surfactant و بيبوظ ال Muco ciliary ،، فده بيخلي ال secretion يحصلها stagnation

لكن «

ال infection هي كا role بتعمل exacerbation
لكن مش هي السبب

وخلي بالك من نقطة مهمة «

إن ال chronic bronchitis مكنتش acute وبعدين بقت chronic
لا هي chronic disease from the start وطبيعته إنها Inflammatory disease
وأهم سبب التدخين
ودور ال infection مجرد exacerbation
وأكثر اثنين organisms؟؟؟ ال Hemophilus و ال pneumo-cocci

ال pathology

• بيكون فيه simple bronchitis الي هي ال early stage ودي بتبقا مجرد mucus secretion كتيرة
بعد فترة ال Mucus الكثير وال cilia الي باظت وال surfactant والكلام ده كله بيتندي المريض يبقا Liable to recurrent
infection وتبقي muco purulent stage

• المرحلة الي بعدها بيحصل air way obstruction كامل لأن ده chronic irritation, chronic inflammation
وبالتالي ال bronchial tree بتبقا مليانة collagen fibers بقا وتببقا obstructive تماماً

المرحلة الأولى يمكن تكاد تكون reversible لو الواحد اتعرف بدري يعني
لكن بعد كده الموضوع almost irreversible

طبعا ال COPD هو mainly clinical case
في الامتحان لو هيناقشك يعنيهيناقشك في الكلام ال clinical

ال clinical pictures

عادة بيكون male « و عادة هذا ال male بيكون فوق الأربعين سنة مدخن وبishtكي شكوى بسيطة
chronic productive cough «، وال sputum أيضاً small amount «، ويبقا whitish لأنه مجرد mucous مفيش
odor ولا أي حاجة خالص «، بس بيزيد الصبح أكثر زي ما احنا عارفين
طب المريض ده هل ممكن ينهج ؟؟؟؟ أه ممكن ينهج لو advanced case
لو دخل في ال emphysema ودي بتبقى Late يعني
ممكن ينهج بدري لو حصل element of broncho spasm ممكن طبعا ينهج
طب هذا المريض بالنسبة لحتة ال exacerbation بتاعته دي نقطة مهمة جدااا
أهم سبب بيعمله exacerbation زي ما احنا اتفقنا ال chest infection

وممكن يجيبك problem يقولك مريض مدخن وسنه ستين سنة وعنده cough و sputum الصبح والكلام ده كله
ثم بيتندي يقولك جاله chest infection

طب ال chest infection إيه العلامة بتاعتها ؟؟؟

العيان أصلاً بيكح وأصلاً بيحبب sputum فاحنا اتفقنا : ال fever ممكن تكون علامة من علامات ال chest infection
بس مش شرط في كل الحالات

مجرد إن ال sputum زاد في الكمية ، مجرد إن ال sputum بقا discolored " اتغير في لونه "
مجرد إن ال sputum بقا الصبح ولبيل دي إشارة إلى إنه فيه Infection

يبقا إنت هنا لا بد أن تُبادر سريعاً بعلاج ال Infection ، و إلا هتبص تلاقي المريض بدأ يتدهور ، وبدأ يفقد الوعي ، وبدأ يدخل في respiratory failure ودي شريحة كبيرة من العيانيين في المستشفيات
ناس عندهم COPD وعندهم دايما Inter current infection
وكل ما يجيله chest infection ممكن يخش ببساطة في respiratory failure الحتة دي مهمة اووووي

Examination نيجي بقا نشوف ال

ال **general examination** هنلاقيه ببساطة ممكن عنده شوية puffiness من كتر الكحة
ال cyanosis لما تبقا الحالة advanced

ال clubbing بيكون Unusual في ال COPD
ال COPD ، may lead to clubbing ،

ممكن بس مش common

ال common لو ال clubbing حصل في ال COPD يبقا الراجل ده دخل في bronchogenic carcinoma أو دخل في
brochiectasis

بردو الناس دي هيجلهم enlarged tender liver , lower limb edema , congested neck vein
دي علامات ال Cor pulmonale الي هي Right sided heart failure
ال epigastric pulsations كل دي علامات

ال pulse خلوا بالك في ال chest disease بيكون فيه دايماً CO2 retention وال CO2 retention بيعمل
vasodilatation يديني ال Big pulse volume من الحاجات المشهورة

لما نيجي تشوف العيان clinically بتلاقي ال chest بيكون ال symmetrical
وبتلاقي ال antero-posterior diameter ممكن يزيد شوية باعتبار إنه هيخش في emphysema
ولما تيجي تشوف ال palpation هتلاقي ال TVF هتلاقيه equal on both sides وهتلاقيه كله resonant ومفيش هنا أي
dullness بالعكس فيه هنا hyper resonance لو دخل في emphysema

auscultation أما ال

ال breath sounds بيكون harsh vesicular breathing يعني ال expiration prolonged
طب ال expiration هنا أصبح prolonged زي ما اتفقنا ، لأن ال expiration أصبح active نتيجة ال air way
obstruction الذي يحدث

يعني المريض وهو بيعمل expiration بيحس أنه فيه resistance في خروج الهواء ، فهو غصب عنه بيحول ال passive
expiration إلى active expiration النتيجة إنه يبقا harsh vesicular breathing
بالنسبة لل rhonchi

ممكن يكون فيه rhonchi نوعين ال broncho spasm هتبقا هنا ال rhonchi هتكون generalized وهتبقا sibilent
أكثر....أهم حاجة إنها Persistent after cough
لما نيجي نبص على ال rhonchi بتاعت ال secretion دي بردو وضع قائم

بتلاقي scattered ، تحت كده حنة حنتين في صدره وبعدين تيجي تقوله كُج ...
يكح تختفي أو تتغير تماماً يبقى ده غالبا secretion

هل ممكن أسمع crepitating ؟؟؟ أه ممكن بالذات في ال secretion
هتكون coarse crepitation ، يعني واحدة كل ال Inspiration ، وهتبقا أحياناً تأخذ جزء من ال expiration
وهتكون low pitched وبتكون low frequency
بس هنا non consonating مفيش هنا وسط مكبر يعمل أي consonating
دي تقريبا الحاجات بتاعت ال Local examination

حنة ال **complications of COPD** أو ال complications of chronic bronchitis
دي ممكن تكون **سؤال** فرضنا إنه حَب يجيبك مسألة COPD واحد بيدخن وكده
وبعدين يقعد يتكلم عن ال respiratory failure وبعدين يقولك إيه ال complications اللي ممكن تحصل لهذا المريض
أهم ال complications طبعا إنه هيخش في respiratory failure وطبعاً ده بيكون type2
اللي هو hypoxic hypercapnea زي ما هنشوف بعد شوية إن شاء الله
ممكن يخش في bronchogenic carcinoma لأن التدخين هنا هو أساس المشكلة

يُقال إن الناس دول أحياناً بجيلهم peptic ulcers طبعا هذا الانسان originally مدخن
ثانياً ال hypoxia and hyper capnea يقولك : إنها بتأثر على ال viability of gastric mucosa طبعا لو خد كورتيزونات
وحاجات من دي بردو بتكون irritant بردو
وال aminophylline منقدرش نقول عليه Irritant أووي لدرجة ال Peptic ulcer يعني

يبقا هنا ال corticosteroid ، smoking ، hypoxia and hypercapnia وأيضا ال
طبعا المريض ده خلوا بالك ال COPD بطبيعة الحال بيعمل polycythemia
يعني دائماً ال COPD بيعمل polycythemia
السؤال هنا **إمتى ال COPD يدخل المريض او يحصله انيميا ؟؟؟** لو بدأ يخش في bronchiectasis
فيبتدي يخش في anemia of chronic disease ، أو العيان جاله بقا على سبيل المثال
ال peptic ulcer وحصل منها bleeding ولا حاجة

طبعا ال **investigations**
ECG ، **X-ray**
ال ECG هيجيب ال right ventricular enlargment بتاع ال cor-pulmonale
ممكن ال cor-pulmonale يعلمي right ventricular enlargment وساعات right axis deviation
طبعا ال cor-pulmonale مش بالمية بالمية بتشخص بال ECG لومبانش ال ECG هيبان ال ECHO
خلي بالك العيان عنده emphysema فانا لما أحط على صدر المريض رسم القلب
ممكن ميبانش أووي من كتر ال emphysema اللي عنده
حتى ال emphysema لو تفتكروا بتعمل لفظ كده في ال ECG ؟؟؟ Low voltage يعني بتخلي ال complexes كلها صغيرة
يقوم ميبنش كويس ال right ventricular enlargement هنا بقا يبينه ال ECHO
بعد كده فيه شوية ال blood examination طبعا ال **blood gases** هتبقا normal في الأول ،

لكن ممكن يبقا فيه hypoxia hypercapnia لو حصل respiratory failure اللي هو غالبا بيكون type 2

العلاج بقا

الأول بتعمل bronchial drainage بايه؟؟؟

عن طريق ال mucolytic expectorants , broncho dilators

يعني فتح ال bronchial tree وخرج

طب إيه هم ال bronchodilator؟؟؟ هتلاقي ال aminophylline وال Beta 2 agonist وال Anti cholinergic زي

ipratropium bromide

ودايمًا إحنا نحب أوووي ال inhaler حتى الناس اللي عندهم COPD ممكن يأخذ Inhaler

يعني مش كل واحد بياخد بخاخة لازم يكون 100% bronchial asthma يعني

ممكن يكون COPD عادي يعني

الكورتيزون عليه خلاف لأن طبيعة المرض قولنا ... inflammatory

طب ما ال anti inflammatory steroid ما أنا أدبه؟؟؟؟!!!!

قالك : لا لالالا

لو إنت عايز تديه ،، لو إنت شايف المريض عنده severe bronchospasm بالرغم من ال broncho dilators اللي بياخذها

عمالين ندليه B2 against والراجل صدره بيزيق وتعبان

أو الراجل عنده allergic element جامد

فأنا خلاص هاضطر أعمل shifting to steroid بس بشرط إن أنا أتابعه يعني ...

لو لقيت الوظائف بتاعت التنفس بتتحسن مع الكورتيزون يبقا أنا باخد ال benefit

لو مفيش بقا تحسن يبقا لا داعي إني أعرض نفسي لل steroid

طبعاً زي ما اتفقنا ان ال B2 agonist لما تفكر فيها وال steroid لما نفكر فيها نفكر في ال inhaler الأول

الأول لما تلاقى ال Inhaler ميجيش زي ما بنشوف في ال bronchial asthma بنوصل لحد step 4

في الآخر عشان أنا ابتدي أدبي هذه ال systemic medications

طب وبعدين هنا العيان هيجيله Intercurrent infection

يعني هنديله anti biotic والانتبي بيوتك مثلاً ممكن نديله amoxacilline , ampicillin ،،

لأن ال organism هنا H.influenza or pneumococci

العيان ده chronic chest disease بيتدهور تماماً لما بجيله chest infection

سواء لما بجيله viral or bacterial

طب ما نديله pneumococcal vaccine؟؟

ال influenza vaccine ال H.influenza vaccine كل دي vaccines بقت موجودة يعني المفروض

لو فيكم واحد باباه عنده advanced COPD أوووي

take care أوووي عشان ال chest infection لوحدها تضيع العيان

خلي بالك وتفضل معاه على هذا المنوال

لو عندك قدرة بقا تعمل transplantation يعني فيه حالات

فيه حاجة اسمها treatment of exacerbation of COPD

أنا إزاي أعالجه ؟؟؟ أنا عموماً أي مريض عنده chest infection وهو chronic bronchitis واتهوور أنا ممكن اديله antibiotics و bronchodilator وال steroid طبعا هتلاقي مكتوب في الكتب الي بتذاكر منها ال diuretics ال diuretics هنا عشان ال right sided heart failure لأن العيان هنا لما بيتدهور جداااا لو هو border line right sided heart failure ال right sided failure بيتدي يتزايد على المريض ال exacerbation معناها إنه more hypoxia وال more hypoxia معناها more pulmonary hyper tension لأن ال hypoxia بتعمل حاجتين

- أول حاجة pulmonary vasoconstriction
- وتاني حاجة هي polycythemia تتزايد

هتلاقي ال pulmonary pressure بيرتفع أحسن حاجة لل pulmonary pressure وفي نفس الوقت أحسن حاجة لل right sided failure عشان يستريح هي ال diuretics ببساطة

ال Emphysema

لازم ال **definition** يبقى في دماغك اللي هو abnormal dilatation لل air spaces ودي distal to terminal bronchioles ال emphysema لازم تبقا واحد بالك إنها with rupture of the inter alveolar septum لأن فيه حالة شبه ال emphysema الي احنا بنسميها **hyper inflation** وال hyper inflation ده بيبقا موجود في حالات ال bronchial asthma على سبيل المثال الهواء بيخش مبيطلعش ،، الهواء بيخش مبيطلعش ،، فتلاقي ال alveoli وكل حاجة distended بس مفيش rupture of inter alveolar septa

دي حته فيه بعض ناس بيقدوا يسألوا فيها معرفش ليه !!!! ... يعني إنت يا دكتور لما تقعد تسألني في حاجة زي كده بتسألني إني افرق بينهم clinical ما هو صعب إني أفرق بينهم clinical الصراحة ال definition أه اقولك ماشي أقدر أفرق بينهم إن ال hyper inflation زي ال emphysema بس مفيش rupture of the inter alveolar septa

أسباب ال emphysema

فيه ال senile ،، وفيه اللي هي compensatory << وطبعا ال compensatory emphysema زي واحد عنده مثلا unilateral lung disease يصح إن ال Lung الثانية تكون hyper inflated بس هما حبوا إنهم يقولوا على ال compensatory emphysema قول عليها أفضل compensatory hyper inflation

طبعا فيه نوع من ال emphysema مهم جدااااا إننا نكون عارفينه اللي هو ال alpha one anti trypsin deficiency ال alpha one anti trypsin deficiency كلنا عارفين إن ال alveoli فيها proteases و anti proteases

ال proteases دي عبارة عن enzymes
 بتحاول زي ال collagenase ، بتحاول إنها digest ال alveolar wall
 فربنا خلقلهم ال anti proteases الي هو على رأسهم alpha one anti trypsin
 يعني الي عنده alpha one anti trypsin deficiency هيبقا عنده مشكلة
 هيبقا عند ال proteases هتبقا dominant على ال anti proteases
 ويبتدي ال alveolar wall يتأكل مع مرور الزمن ويجي على سن الأربعين سنة
 يعني خلي بالك ال congenital emphysema دي بيتجي على سن الاربعين سنة أو أكثر
 يعني ممكن تكون بعد الاربعين كمان
 وانا بشخصها كمان بال exclusion ألاقى مريض عندي عنده emphysema
 ومعدوش أي حاجة من الحاجات ال COPD الي احنا عارفينها

بالنسبة لحتة ال smoking هنا وعلاقته بال emphysema
 زي ما ال smoking عمل inflammation in bronchial tree
 هذا ال inflammation في ال Lung وهذا ال chronic irritation بيخلي كمية ال neutrophils جوا ال lungs كثيرة جدا
 حتى إنه يقولك : Increased ploymorph influx
 جوا ال lung يعني نفس التدخين وحاجة irritant تخلي الرئة بتاعتنا مليانة على عينها macrophages
 مليانة neutrophils فتلاقي ال proteases تطلع من هذه ال neutrophils بكميات كبيرة جدا
 عشان كده ال smoking نفسه هو بيعمل air way obstruction in bronchial tree
 وبيخلي ال proteases تتزايد directly ،، عشان كده ال smoking بيعمل emphysema برود
 لأنه كان زمان بيقولك ال smoking بيعمل chronic bronchitis وال chronic bronchitis بتعمل emphysema
لالالالالالالال
 هي ال chronic bronchitis بتعمل emphysema عن طريق ال partial bronchial obstruction الهواء يخش ميطلعش
 فال air way تبقا distended ده mechanical factor
 هناك Inflammatory factor من ال smoking نفسه بقا
 ال cytokines وال proteases بتطلع من ال neutrophils وحاجات
 فالماضيع كلها عشان كده التدخين بيعمل chronic bronchitis و emphysema
 وال process ماشية مع بعضها
 لكن مفيش شك إن ال chronic bronchitis ليها factor mechanical ،، الي هي ممكن تعمل برود emphysema نتيجة
 ال trapping of air

طبعاً ال clinical pictures

بتاع مريض ال emphysema هيكون عنده شوية dyspnea
 بس ممكن برود يديني history واضح ل COPDيبقا الحالة COPD ؟؟؟ لا ده المريض ده معدوش COPD وسنه مش كبير
 طب إيه الي يعمل ال emphysema كده ؟؟؟
 ساعات بنجيها by exclusion وتكون alpha one anti trypsin deficiency
 خلي بالك ال alpha one anti trypsin deficiency دي مشهورة بحاجة ،، إنها بتبقا معاها أحيانا liver cirrhosis
 خلي بالك يعني هنا فيه علاقة بين ال Liver وال Lung
 إيه المرض الي جمع ال Liver cirrhosis بال emphysema ؟؟؟

ال alpha one anti trypsin deficiency متنساش

طب إيه ال **Investigations**
 كل اللي هلاقيها أعمل **chest X-ray** هلاقي ال lung هتكون hyper inflated
 وهتلاقي ال heart لو فاكرين هيكون heart ribbon shaped طويل كده
 وبعدين أنا لو جيت عملت investigations زي ال **ECG** متنساش هتلاقي ال ECHO low voltage
 هيجيب هنا ال cor-pulmonal أفضل
 ومتنساش بردو **alpha one anti trypsin assessment**
 طبعا ال **blood gases** هتلاقي hypoxia hyper capnia لو دخل في respiratory failure

والعلاج non specific
 العلاج إنت بتعالج ال COPD نفسه اللي هو Originally موجود
 لو إنت أثبت إنه فيه alpha one anti trypsin deficiency
 يبقى إنت راجل يعني محترم وشخصت المرض ده ،، ويا ريت تشخصه بدري وتدي alpha one anti trypsin injection early

فيه حنة قدمة اسمها ال pink puffer وال blue bloater خد عنها فكرة
 حالات ال COPD هي عبارة عن chronic bronchitis و emphysema
 فهو السؤال هنا بيجي لو حب يقولك : يعني مين ال dominant على الثاني
 ال chronic bronchitis ولا emphysema ؟؟؟؟

أنا هاتكلم الأول عن ال blue bloater عشان تفهم ال pink puffer
 ال **blue bloater**
 ال blue يعني cyanosis ال bloater يعني منفخ يعني عنده edema يبقى بالبلدي كده
 كل ما كان المريض cyanosed وكلما كان المريض right sided failure و edema
 يبقى هنسميه blue bloater ،،، هنا معناها إنه ال chronic bronchitis هي ال dominant
 جت منين ؟؟؟؟

هو القصة إن البني آدم اللي عنده chronic bronchitis dominant إيه مشكلته ؟؟؟؟
 إن عنده ال air way ضيق ال air way نفسه ضيق
 فالراجل بيعاني من obstructive hypo ventilation يقوم يحصله hypoxia
 طب هو يعوضها بايه ؟؟؟

إنه يعمل hyper ventilation compensatory
 السؤال هنا هل هيقدر ولا مش هيقدر ؟؟؟
 خلي بالك هو عنده obstructive hypo ventilation
 يعني ال air way ضيق ،،، يعني هيحاول يعمل hyper ventilation مش هيعرف ☹
 ليه ؟؟ لأن ال air way already ضيق فلن ينجح في عمل ال hyper ventilation أبداً
 فيظل المريض hypoxic على الآخر
 ال hypoxia هتعمل cyanosis هتعمل vasoconstriction in pulmonary arterioles
 والنتيجة pulmonary hyper tension و right sided failure

دا اللي كانوا بيقلوا عليه blue bloater
يعني كان عنده Hypo ventilation من ال obstruction
ومش هيعرف يعمل compensatory hyper ventilation
هيعمل ازاي وهو عنده obstructive ventilation defect !!!!

لكن الثاني اللي هو ال pink puffer
ده ال emphysema هي ال dominant
ال emphysema بتعمل hypoxia
ولكن ال hypoxia بتاعت ال emphysema هنا نتيجة إنه فيه alveoli بيحصلها rupture
وفيه alveoli زي ما تقول مبقتش healthy عشان تعمل gas diffusion مع ال blood capillaries
فتلاقي ال emphysema فيها شوية Hypoxia
ويقال انها mild فالمرضى بيعوض ال hypoxia بانه يعمل hyper ventilation
فيجي يقولك هو ال emphysema المشكلة اللي فيها إيه؟؟؟
هي فيها مشكلة diffusion defect ان ال alveoli مش healthy أوووي
فتلاقي ال exchange بتاع ال gases مش اوي
دي بتتعرض بإيه بقا؟؟؟
إن ال alveoli اللي لسه healthy بتكون لسه hyper inflated كويس بالهواء
فالمرضى بيتدي يعمل hyper ventilation يأخذ نفس جامد
فال hyper ventilation دي بتعوض شوية ال hypoxia اللي عند المريض
فالمرضى ميقاش Hypoxic أوووي
يقوم المريض ميزرقش ولا يورم فيبقا لونه ال hypoxia البسيطة
متزقش بالعكس بتعمل polycythemia فيبان وشه محمر عشان كده بنقول عليها pink

كلمة puffer بقا يعني هو بيخرج ال expired air من خلال زي ما قولنا بؤه ويبقا ضامم شفائفه بنسميها pursed lips بيه؟؟؟
عشان ال alveoli وال interstitial tissue فيها هواء كتير
خلي بالك اللي عنده emphysema لعملك لما تيجي في ال pathology تلاقي انه ال emphysema عاملة مشكلة
عاملة زي ما تقول كده high pressure جوال ال alveoli و high pressure في ال interstitium tissue
فدول بيضغطوا على ال small bronchiole الصغيرة فممكن يقفلوها
عشان كده الراجل بيحاول to keep small bronchioles patent
يقوم يخرج ال expired air بتاعه في شكل متدرج
الشكل المتدرج ده بيخلي ال small bronchiole بتاعته متبقاش obstructed rapidly و collapsed
بال high pressure اللي في ال surrounding alveoli وال interstitium tissue
فالمرضى بيبان إنه بيخرج نفسه وهو ضامم بؤه والهواء يخرج متدرج عشان يفضل ال intra bronchial pressure high
يقوم ال bronchiole الصغيرة متبقاش obstructed ولا collapsed
بص محدش هيسألك اوي في الكلام ده بس جازي بردو ☺

فيه حنة مكتوبة يقولك clinical criteria of severe COPD

إليه اللي يقولك ان ال COPD ده severe ???

١. والله هأقولك المريض عنده increase of the antero-posterior diameter ده رقم واحد .
 ٢. عنده ال accessory muscles شغالة ال sterno mastoid والترايزيس والكلام ده كله.
 ٣. عنده كمان positive litten sign (Inter costal suction (indrwaing during inspiration) الي هي
 ٤. عنده كمان حاجة اسمها expiratory filling of neck vein .
- دي كلها علامات تقولك ان الراجل ده حالته severe case of COPD

Suppurative Lung Syndrome (SLS)

Definition

- Group of diseases characterized by cough and expectoration (Big, Purulent, Postural, foeted بريحة and Paroxysmal).
- These diseases are:
 - Lung abscess.
 - Bronchiectasis.
 - Infected lung cyst.
 - Empyema with bronchopleural fistula.

Lung abscess

Aetiology

i. Primary lung abscess (Inhalation) : "common"

- Inhalation of septic material: e.g. upper respiratory secretions, vomitus or foreign body.
- Absence of respiratory defense mechanisms e.g. cough reflex.
- Anaesthesia, coma or convulsions.
- Causative organisms include :
 - Anaerobes e.g. Bacteroides.
 - Gram-positive cocci e.g. Staph aureus.
 - Gram-negative bacilli e.g. Klebsiella.

ii. Secondary lung abscess:

1. Lung disease:

Pneumonia: especially staphylococcal & Friedlander's pneumonias, Bronchial carcinoma, Bronchiectasis, lung collapse, pulmonary infarction & lung cyst.
2. Subdiaphragmatic disease:

Amoebic liver abscess, subphrenic abscess.

3. *Mediastinal disease:*
e.g. Cancer oesophagus.
4. *Chest wall disease:*
Penetrating wounds, Osteomyelitis of vertebrae, ribs or sternum.
5. *Pyaemia* : (Pyaemic abscesses)
May occur due to e.g. infective endocarditis, septic thrombophlebitis, osteomyelitis (septic focus).
 - *The causative organism is usually Staph Aureus.*

Complications

1. Chest complications

- Severe haemoptysis.
- Spread of infection to other parts of the lung leading to recurrent pneumonia & abscesses.
- Fibrosis & bronchiectasis around the abscess cavity.
- Pleurisy, effusion, empyema, pyopneumothorax & pleural fibrosis.

2. General complications

- Metastatic abscessed especially in the brain.
- Anaemia & amyloidosis in chronic cases.

N.B. Signs of a cavity are variable depending on :

- **Site:** superficial or deep.
- **Size:** small or large.
- **Surroundings:** consolidation or fibrosis.
- **Draining bronchus:** patent or occluded.
- **Contents:** full or empty.

N.B. Signs of a cavity include:

- **Inspection:**
 - The shape is usually normal. Rarely retraction occurs if there is marked fibrosis.
 - Limitation of respiratory movements on the affected side.
- **Palpation:**
 - The mediastinum is usually central.
 - Rarely shift to the same side occurs if there is marked fibrosis.
 - TVF is increased only if the cavity is superficial & surrounded by consolidation.

➤ **Percussion:**

- Localized dullness over the site of the abscess.

➤ **Auscultation :**

- Bronchial breathing: cavernous or amphoric type.
- Crepitations: may be medium-sized or coarse, consonating or non-consonating.
- Bronchophony, aegophony & whispering pectorilology may be present.
- Post-tussive suction may be heard over a collapsible cavity.

Investigation

1. Chest X-ray

- Cavity with fluid level.
- Features of the causes or complication.

2. Sputum

- Culture & sensitivity.

3. Chest CT scan

- Diagnostic.

4. Bronchoscopy

- Localize the site of the abscess.
- Exclude the presence of bronchial carcinoma.
- Detect & remove foreign body.
- Drain pus & inject antibiotics into the cavity.

5. Bronchography

- indicated mainly to exclude bronchiectasis.

Treatment (4)

1. General

- Rest.
- Plenty of fluid.
- Nutrient diet.

2. Symptomatic treatment

- **Mucolytics** e.g. bromhexine hydrochloride.
- **Expectorants** e.g. potassium iodide.
- **Analgesic**: Pain killers.
- **Antipyretic**: NSAIDs.

3. Specific**A. Drainage of pus**1. Postural drainage

2-3 times daily – the patient usually lies on the healthy side.

It may be helped by percussion – associated with respiratory exercises.

2. Bronchoscopic aspiration: with injection of antibiotics is rarely used.3. External drainage: is rarely used.**B. Antibiotic therapy:**

Choice of antibiotics depends on culture & sensitivity tests.

The most commonly used drugs are:

- Clindamycin or metronidazole for Staph. Aureus.
- Aminoglycosides or cephalosporins for Klebsiella (Gram negative).

C. Surgical treatment:

Segmentectomy or lobectomy may be indicated in the following conditions:

1. Failure of medical treatment.
2. Complicated: Severe recurrent haemoptysis, empyema or pyopneumothorax.
3. Severe
4. Association: Surrounding bronchiectasis or suspension of malignancies.

4. Treatment of complications**BRONCHIECTASIS****Definition**

Abnormal permanent dilatation of terminal bronchi

1. Congenital bronchiectasis (2)1. *Primary* : Isolated bronchiectasis.1. *Secondary*:

a. Immotile cilia syndrome

- Bronchiectasis.
- Sinusitis & otitis media.
- Male sterility.
- May be associated with dextrocardia & situs inversus totalis (Kartagener's syndrome).

b. Cystic fibrosis.

c. Immunodeficiency syndromes

2. Acquired bronchiectasis (2)**1. Infection and Fibrosis :**

- a. Destruction of the bronchial muscles & elastic fibers resulting in bronchial dilatation.
- b. Peribronchial fibrosis causes traction over the bronchi leading to further dilatation.
- c. (As, broncopneumonia, whooping cough, measles, TB & lung abscess especially during childhood).

2. Bronchial obstruction:

- a. Partial obstruction : valve-like mechanism
- b. Complete obstruction.

Pathology

- **Site:** bilateral & basal, But may be apical in TB or localized in foreign body or tumor.
- **Shape:** bronchi may be cylindrical, tubular, fusiform or serpentine.
- **Changes:** bronchi are dilated with desquamation & ulceration of the mucous membrane.
- **Others:** The surrounding lung tissue may show consolidation, Emphysema of the upper parts of the lungs.

Complications (As L. Abscess)**Clinical picture****Symptoms****1. Suppurative syndrome:**

- Paroxysmal cough related to posture with expectoration of excessive purulent foeted sputum.
- More common in the morning and on stooping foreword or lying down.
- The onset is usually insidious & the course is progressive over years with winter exacerbations.

2. Haemoptysis:

Usually blood- tinged sputum but frank haemoptysis may occur.
(bronchieetasis sicca haemorrhagica) secondary to TB.

3. Dyspnea & Chest pain**4. Symptoms of complications****5. Sinusitis:** is a common association

Signs**General signs**

- Recurrent fever.
- Pallor & loss of weight.
- Puffiness of eye lids due to chronic cough.
- Clubbing of fingers & may be hypertrophic osteoarthropathy.
- Oedema of lower limbs may be present.

Chest signs (Bronchiectasis is usually bilateral & basal)

- **Inspection:**
 - Normal shape of the chest Rarely retraction occurs if there is marked fibrosis.
 - Diminished respiratory movements mainly in the basal parts.
- **Palpation:**
 - The mediastinum is central.
 - TVF may be increased on the basal parts.
- **Percussion :**
 - Patchy dullness on the basal parts.
- **Auscultation:**
 - Areas of bronchial breathing & crepitations on the basal parts.
 - Crepitations may be medium-sized or coarse, consonating or non-consonating.
 - VR : increased.

Investigation**1. Chest X-ray**

- Ring shadows which may give honey-comb appearance usually in the basal parts.
- Parallel lines which may give honey-comb appearance usually in the basal parts.
- Parallel lines which may give tram-lines appearance.
- May show hypertranslucency in upper parts due to emphysema.

2. Sputum culture & sensitivity test

- The most commonly isolated organisms are H. influenza & St. pneumoniae.

3. CT chest

- For accurate diagnosis & localization.

4. Bronchography

- The investigation of choice to confirm the diagnosis and to localize the site, extent of bronchiectasis before surgery.
- **Bronchiectasis** appears as cylindrical, saccular, fusiform or varicose dilatation of the **bronchi**.

5. Bronchoscopy

- May reveal the underlying cause.
- May be used for drainage of pus & injection of antibiotics.

6. Investigations for diagnosis of the cause

- e.g. tests for cystic fibrosis.

Differential diagnosis

- *Causes of suppurative syndrome.*
- *Causes of haemoptysis.*

Treatment (4)**1. General**

- Rest.
- Plenty of fluid.
- Nutrient diet.

2. Symptomatic treatment

- **Mucolytics** e.g. bromhexine hydrochloride.
- **Expectorants** e.g. potassium iodide.
- **Antipyretic**: NSAIDs.
- **Analgesic**: Pain killers.

3. Specific**A. Drainage of pus****1. Postural drainage**

2-3 times daily – the patient usually lies on the healthy side.

It may be helped by percussion – associated with respiratory exercises.

2. Bronchoscopic aspiration: with injection of antibiotics is rarely used.**B. Antibiotic therapy**

Choice of antibiotics depends on culture & sensitivity tests.

The most commonly used drugs are:

- Clindamycin or metronidazole for anaerobes.
- Cloxacillin or vancomycin for Staph. Aureus.
- Aminoglycosides or cephalosporins for Klebsiella (Gram negative).

C. Surgical treatment:

May be indicated in the following conditions:-

1. **Failure** of medical treatment.
2. **Complicated**: Severe recurrent haemoptysis, empyema or pyopneumothorax.
3. **Severe**.
4. **Association**: Surrounding Abscess or suspension of malignancies.

4. Treatment of complications**Cystic fibrosis**

Autosomal recessive disease characterized by :

- Viscid secretions followed by bronchiectasis and super infection by pseudomonas mainly.

Investigations

As bronchiectasis + SWEAT TEST

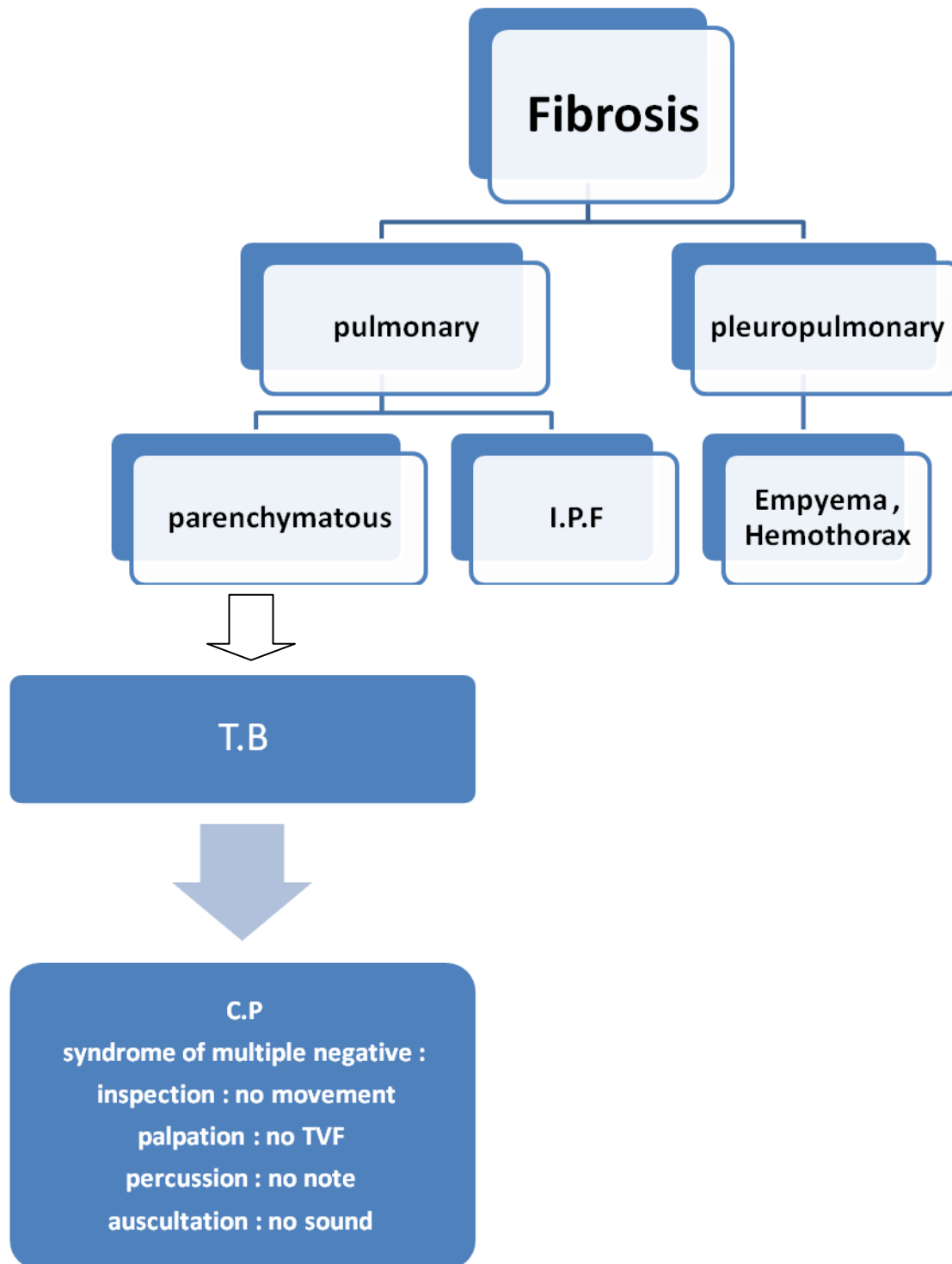
Treatment

As bronchiectasis

N.B.

- *Absent vas deferens is a common association.*

Fibrosis



INTERSTITIAL PULMONARY DISEASE

Aetiology: (3 I, 2C, 2M)

- **Idiopathic pulmonary fibrosis (IPF).**
- **Immune diseases.**
- **Inhalational lung diseases.**
 - Extrinsic allergic alveolitis: (due to inhalation of organic dust) e.g.
 - Farmer's lung.
 - Bagassosis.
 - Bird fancier's disease.
 - Pneumoconiosis: (due to inhalation of inorganic dust) e.g.
 - Silicosis.
 - Asbestosis.
- **Chronic pulmonary diseases (Granulomatous) e.g.**
 - Sarcoidosis.
- **Chronic pulmonary venous congestion.**
- **Familial:**
 - Histiocytosis X.
 - Neurofibromatosis.
- **Malignancies:**
 - Lymphangitis carcinomatosa.
 - Bronchoalveolar carcinoma.
 - Lymphomas & Leukaemias.
- **Miscellaneous**
 - Idiopathic haemosiderosis.
 - Alveolar proteinosis.
 - Alveolar microlithiasis.

Pathophysiology

1. Impaired diffusion.
2. Restrictive hypoventilation.
3. Hypoxia with normo or hypocapnia.

Clinical picture

- Gradual progressive **dyspnea**.
- **C**ough.
- **C**entral Cyanosis.
- **C**lubbing.
- **C**repitations: late or pan-inspiratory medium-sized, non-consonating.
- **C**ause e.g. occupational exposure to dust.
- **C**omplications:
 - Respiratory failure.
 - Pulmonary hypertension & Cor pulmonale
 - Pulmonary infections.

Investigations**1. Chest X-ray**

- Bilateral diffuse affection of lungs with reticulations & micronodular infiltrations (usually basal).

2. Pulmonary function tests:

- Ventilation test.
- **Decreased** lung volumes especially forced vital capacity.
- **Decreased** lung compliance
- Normal expiratory flow rates
- Diffusion tests: Decreased CO transfer factor.
- Arterial blood gases:
 - **Decreased PO_2** .
 - **Decreased PCO** .

3. Investigations for the cause**Differential Diagnosis**

1. Causes of respiratory failure
2. Causes of hypoxic cor-pulmonale.

Treatment

1. Treatment of the cause.
2. If the condition is of unknown aetiology e.g. IPF:
 - Steroids: **prednisone** 1 mg/kg/d.
 - Cytotoxic drugs: e.g. Cyclophosphamide.
3. Symptomatic treatment: e.g. oxygen therapy.
4. Treatment of complications:- Pulmonary infections.
 - Respiratory failure.
 - Heart failure.

Disease activity

- **Bronchoalveolar lavage:**
For detection of type and amount of inflammatory cells.
- **Open lung biopsy:**
(inflammatory cells > fibrosis).

Chest (long case)

A. History

Personal history

Mr. Muhammad Ahmad 74 years old, married and has 3 children, the youngest is 26 years old, he is a waiter, from Imbaba, he is a heavy smoker.

Complaint

Patient is complaining of shortening of breathing and chest wheezes of 3 days duration.

Present history

The condition started 30 years ago by gradual onset and progressive course of **cough**, especially at early morning and winter time, increased by exertion, decreased by rest, associated with **expectoration of sputum** which was; scanty, whitish, thick, not foetid and had no special character. The expectoration used to increase in the early morning and winter time, but: there was no postural variations.

Two years later, the patient started to suffer from exertional **dyspnea**, with gradual onset & progressive course, increased by exertion, decreased by rest, not associated with orthopnea or PND, but: it was associated with persistent **wheeze**.

Patient sought medical advice and was admitted to Demirdash hospital where some investigations were done to him in the form of chest X-ray, sputum analysis which was negative, in addition to the usual routine laboratory work-up.

He received bronchodilators (Aminophylline) by infusion, cortisone, antibiotics (Flumox), oxygen inhalation and doses of bronchodilators by nebulizer when needed. He received this medication for 2 weeks till his condition was stabilized & was then discharged.

His condition partially improved, but he used to suffer from repeated attacks of severe dyspnea and wheezes improved by received his usual regular medication.

Six years later, the patient started to suffer from gradual persistent dull aching **pain** in his right hypochondrium increasing by meals & by exertion associated with **oedema** of his lower limbs but with no ascites. He sought medical advice again where diuretics (Lasix) were added to his usual medication & was advised proper bed rest. The oedema disappeared & the pain partially improved & his condition is stationary as such up till now.

There was no haemoptysis, no cyanosis, no compression symptoms, no symptoms of T.B. toxemia & no symptoms of respiratory failure. There were no symptoms of other systems affection.

Past history

There is no past history of DM, hypertension, T.B., allergy, Bilharziasis. There is no past history of previous operations, or blood transfusion.

Family history

Family history is irrelevant.

Diagnosis

A case of bilateral lung disease most probably COPD due to chronic irritation, the patient compensated and complicated by cor-pulmonale.

B. General examination

- The patient with an average general condition, fully conscious, oriented by time, place and persons, with good mood and memory, co-operative with an average intelligency.
- The patient with an average built for his age, he lies comfortable in bed.
- No pallor, jaundice or cyanosis.
- Respiratory rate
 - ✓ Tachypnea with working accessory respiratory muscles.
- Head examination
 - ✓ Puffiness of the eye lid due to chronic cough.
- Neck examination
 - ✓ Congested neck veins due to: increased intrathoracic pressure, or cor-pulmonale.
- Upper limbs examination
 - ✓ **Pulse:** 80 beats / min., regular, variable volume, equal in both sides, pulsus paradoxicus > 15 mmHg. (marked drop of systolic pulse during inspiration).
 - ✓ **Blood pressure:** 110 / 75 mmHg.
 - ✓ *No hand clubbing.*
- Lower limbs examination
 - ✓ Oedema of lower limbs (cor-pulmonale or less commonly salt and water retention).
- Abdominal examination (not in this patient)
 - ✓ Lower border of the liver may be displaced downwards due to (ptosed liver by flat diaphragm or enlarged liver due to cor-pulmonale).
 - ✓ Umbilical or inguinal hernia may be present as a complication of chronic cough.
 - ✓ No ascites (only if RSHF occurred).
- Cardiovascular examination
 - ✓ Absent apex (emphysema).
 - ✓ Faint heart sounds.

C. Local examination

Inspection

Shape of the chest

- Barrel shaped chest (emphysematous) with increased anteroposterior diameter.
- Relatively low-lying diaphragm.
- No scars, dilated veins or pigmentation.
- There is epigastric pulsation & left para sternal pulsation. (right ventricle enlargement).

Respiratory movement

- Diminished bilaterally.
- Mainly abdominal respiration.
- Suction of supra clavicular , supra sternal & lower intercostals spaces during inspiration and fullness during expiration.

Palpation (5 T)

- Central **t**rachea.
- No **t**enderness.
- **T.V.F.:** equal on both sides (diminished all over the chest).
- There is palpable **wheezes** with deep respiration.
- Bilateral limitation of chest **e**xpansion.

Percussion

- Hyper-resonance all over the chest.
- Encroachment on the normal hepatic & cardiac dullness.

Auscultation

- Diminished air entry.
- Vesicular breath sounds with prolonged expiration.
- Generalized wheezes (diffuse, mainly expiratory, polyphonic, changes with cough).
- Crepitation: early inspiratory (opening snap of the chest).
- No vocal resonance.

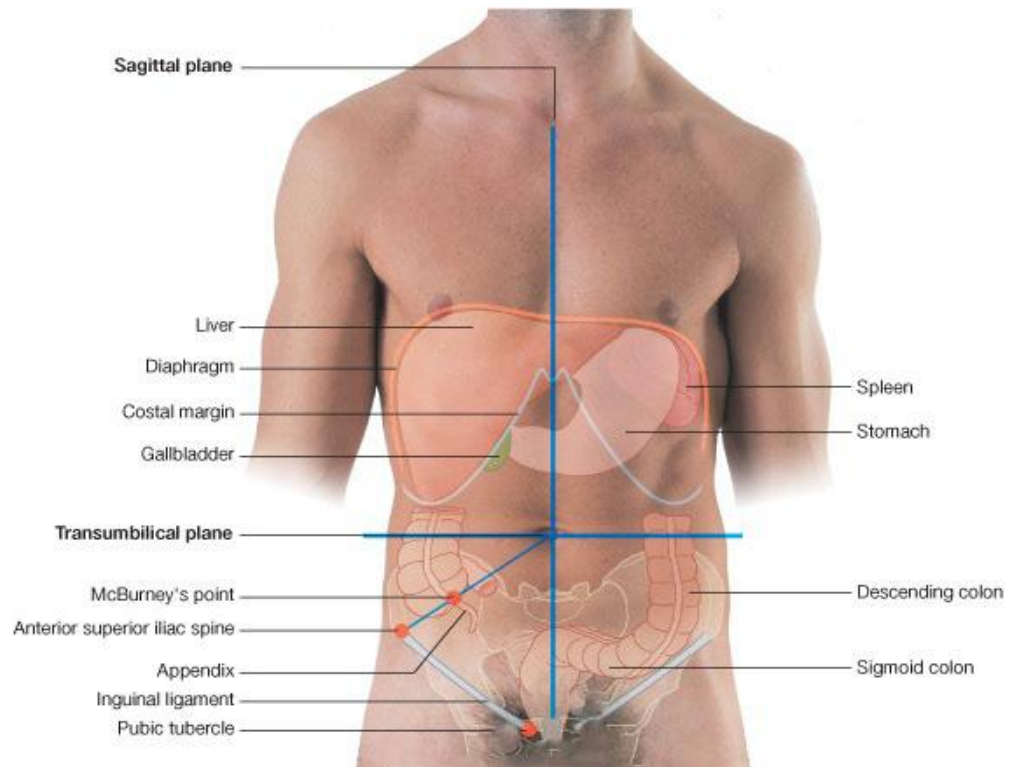
Chest diseases

	Inspection		Palpation		Percussion	Auscultation		
	shape		Mediastinum	T.V.F.		Breath S.	Add. S.	Test
Consolidation	Normal	Limitation of respiratory movement	Central	+++	Dull	Bronchial	Crepitations	-
Cavitation	Normal or retracted		Central or pulled	+++	Dull	Bronchial	Crepitations	Post-tussive suction
Collapse	Retracted		Pulled	±	Dull	Diminished	-	-
Fibrosis	Retracted		pulled	---	Dull	Diminished	Crepitations	-
Ch. Bronchitis	Normal		Central	---	Normal	Harsh V.	Rhonchi	-
Emphysema	Barrel		Central	---	Hyper resonant	Harsh V.	Rhonchi	-
Pleurisy	Normal		central	---	normal	Vesicular	Rub	-
Pleural effusion	Bulge		pushed	---	Stony dullness	Diminished	-	-
pneumothorax	Bulge		Pushed	---	Tympanitic	Diminished	-	Coin test
Hydro pneumothorax	Bulge		Pushed	---	Shifting dullness	Diminished	-	Succusion splash

Surface anatomy of the abdomen

The abdomen can be divided into quadrants by a vertical median plane and a horizontal transumbilical plane, which passes through the umbilicus

- The liver and gallbladder are in the right upper quadrant.
- The stomach and spleen are in the left upper quadrant.
- The cecum and appendix are in the right lower quadrant.
- The end of the descending colon and sigmoid colon are in the left lower quadrant.



The level of the diaphragm varies during the breathing cycle. The dome of the diaphragm on the right can reach as high as the fourth costal cartilage during forced expiration.

Most of the liver is under the right dome of the diaphragm and is deep to the lower thoracic wall.

The inferior margin of the liver can be palpated descending below the right costal margin when a patient is asked to inhale deeply.

The upper part of the abdominal cavity projects above the costal margin to the diaphragm and therefore abdominal viscera in this region of the abdomen are protected by the thoracic wall.

A line between the anterior superior iliac spine and the pubic tubercle marks the position of the inguinal ligament, which separates the anterior abdominal wall above from the thigh of the lower limb below.

The iliac crest separates the posterolateral abdominal wall from the gluteal region of the lower limb.

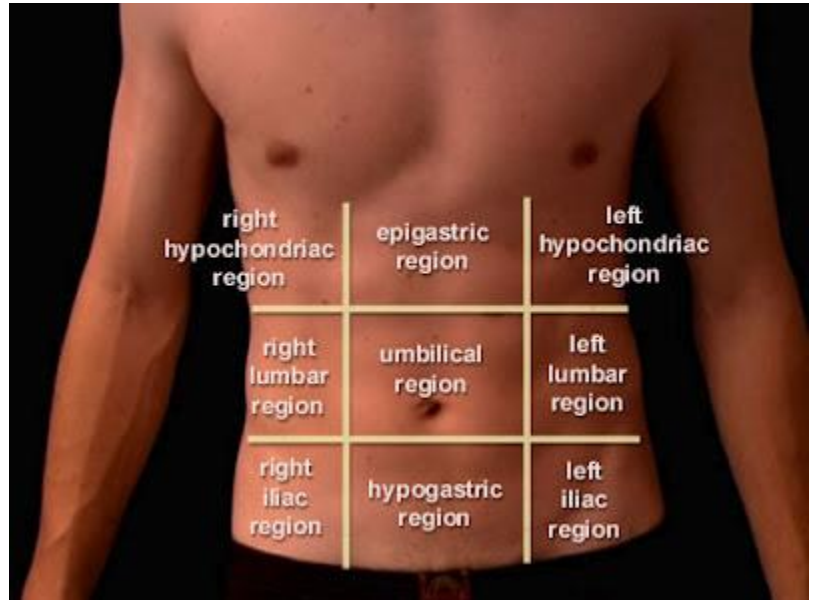
A common surface projection of the appendix is McBurney's point which is one-third of the way up along a line from the right anterior superior iliac spine to the umbilicus.

The abdomen can be divided into nine regions by a midclavicular sagittal plane on each side and by the subcostal and intertubercular planes, which pass through the body transversely. These planes separate the abdomen into:

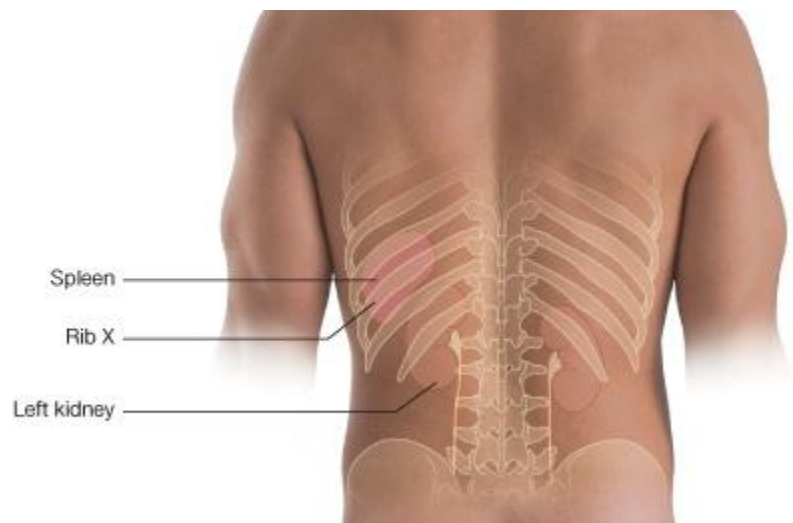
- **three central regions** (epigastric, umbilical, pubic);
- **three regions on each side** (hypochondrium, flank, groin).

N.B.

- Pain from the abdominal part of the foregut is referred to the epigastric region, pain from the midgut is referred to the umbilical region, and pain from the hindgut is referred to the pubic region.



- The spleen projects onto the left side and back in the area of ribs IX to XI.
- The spleen follows the contour of rib X and extends from the superior pole of the left kidney to just posterior to the midaxillary line.



- The kidneys project onto the back on either side of the midline and are related to the lower ribs.
 - the left kidney is a little higher than the right and reaches as high as rib XI;
 - the superior pole of the right kidney reaches only as high as rib XII.
- The lower poles of the kidneys occur around the level of the disc between the LIII and LIV vertebrae. The hila of the kidneys and the beginnings of the ureters are at approximately the LI vertebra.
- The ureters descend vertically anterior to the tips of the transverse processes of the lower lumbar vertebrae and enter the pelvis.

Abdominal sheet

Don't forget talk about

1. History: which include
 - Personal history.
 - Complaint.
 - History of present illness.
 - Past history.
 - Family history.
 - Socioeconomically state.
 - In females (menstrual and obstetric history).
2. Examination :
 - General.
 - Local.
3. Investigations.
4. Treatment.
5. Diagnosis.

History

Personal history

1. **Name**: to be familiar with the patient. اسم حضرتك إيه ؟؟؟
2. **Age**: as certain diseases are more common in certain ages, e.g. عندك كام سنة ؟؟؟
 - Calcular obstructive jaundice : middle aged females.
 - Malignant obstructive jaundice : old males.
 - Cirrhosis: usually in adults.
3. **Sex**
4. **Occupation**: persons in certain occupations are more susceptible to certain diseases e.g.
 - **Farmers**: bilharziasis بتشتغل إيه ؟؟؟
5. **Marital state**: for possible sterility or impotence.

متزوج ولا لسه ؟؟؟ عندك أولاد ؟؟؟ كام ولد ؟؟؟ أصغرهم عنده كام سنة ؟؟؟
6. **Residence**: may reflect socioeconomic condition and may occasionally point to certain disease. e.g.
 - **Country: Bilharziasis** ساكن فين ؟؟؟ طول عمرك ساكن هناك ولا كنت في مكان ثاني قبل كده ؟؟؟
7. **Special habits**: special habit is a habit that makes the patient more susceptible than others to a certain a disease.
 - **Alcoholism in cirrhosis.** بتشرب إيه من الكحوليات ؟؟ والكمية أد إيه ؟؟؟

Complaint

C/O + Duration

إيه اللي جابك المستشفى ؟؟؟

- By the patient own words – No medical terms.
- As short as possible (one complaint is enough).

هنشوف مثال بسيط على كده

نقول مثلاً the patient is complaining of abdominal swelling of **2 weeks** duration

في المثال ذكرنا إن المريض بيشكي من abdominal swelling ، وده من كلام المريض

ومستخدمناش مصطلحات طبية ، وكمان كلام مختصر زي ما أنت شايف

وكمان كتبنا في الآخر ال duration ، اللي كانت two weeks

History of present illness

- i. Analysis of the complaint.
- ii. Symptoms of the related system.
- iii. Other systems.
- iv. Investigations and treatment.

Characterized by:

- As long as possible
- Contains medical terms
- In chronological arrangement
- In the form of a story.

i. Analysis of the complaint**N.B.**

- For analysis any complaint in chest as usual (8) + 3 variants + 3 for any excreta.

For analysis any complaint (8) as usual

- Onset - course - duration.

ال onsets : الشكوى ظهرت فجأة ولا بعد أد إليه ؟؟؟

ال course وال duration : الأعراض ظهرت بالتدريج ولا على مدى أسابيع أو شهور ؟؟؟ أو بتحدث في نوبات

- Association.

هل كانت مصحوبة بحاجة ،، وتذكر الأعراض الثانية التي ممكن تكون موجودة ؟؟؟

- What ↑ and what ↓.

إليه التي يزيد العرض ده ؟؟؟ وإليه التي يقلله ؟؟

- Effect of treatment.

تأثير العلاج عليه ؟؟؟ هل لما بتأخذ العلاج الأعراض بتقل ولا بتفضل زي ما هي ؟؟؟

- Date of last attack.

آخر مرة جالك العرض " وتقول إسم العرض ده " كان إمتى ؟؟؟

For any excreta

- Amount.
- Content - Color - Consistency.
- Odour.

ii. Symptoms of the related system (GBS)

- | | |
|---|--------|
| • <u>G</u> .I.T. (upper & lower). | } 3 Gs |
| • <u>G</u> eneral (toxic). | |
| • <u>G</u> ynecological. | |
| • <u>P</u> ain. | } 3 Ps |
| • <u>B</u> iliary system (hepatobiliary). | |
| • Urinary system. مشاكل في البول | |
| • Abdominal <u>s</u> welling. | } 3 Ss |
| • Lower limb <u>s</u> welling. | |
| • <u>S</u> ystemic review. | |

N.B. oral question in each symptom:

- ✓ definition.
- ✓ Analysis.
- ✓ Causes.

1. G.I.T.

Upper G.I.T.	Lower G.I.T.
➤ Appetite. شهيتك على الأكل اتغيرت ؟؟؟ • Anorexia / polyphagia. ➤ Dysphagia. هل هناك صعوبة في البلع ؟؟ للسوائل أو للأكل ؟؟؟ ➤ Vomiting. حصلك ترجيع ؟؟؟ ➤ Haematemesis. رجعت دم قبل كده ؟؟؟ ➤ Water brush. بتحس باللعاب مالي فمك ؟ ونفسك غمة عليك ؟؟ ➤ Halitosis. رائحة فمك متغيرة ؟؟؟ ➤ Heart burn: in GERD. هل عندك حمو على فم المعدة ؟؟؟ ➤ Eructation. بتتكرع ؟؟؟ ➤ Hiccough (idopathic / CRF). بجيلك زغطة كثير ؟؟؟	➤ Flatulence. بطنك اتنفخت ؟ وفيه غازات ؟؟ ➤ Audible borborygmi. ➤ Bowel habit. ➤ Haematochezia. فيه دم أحمر مع التبرز ؟؟ ➤ Melena. هل هناك براز أسود زي الزيت ؟؟؟

D.D. of Halitosis ?

1. Foeter hepaticus: liver cell failure.
2. Amoniacal: renal failure.
3. Acceton: D.K.A.
4. Foetoid: S.L.S.
5. Local: dental caries.
6. Dietyery.

1. Haematemesis

Preceded	Attack	Followed by
Vomiting	➤ Analysis as above "8 as usual + for any excreta". ➤ History of B.T. = severe attack.	Melena Dark, tarry stool

D.D. with haemoptysis.

يسأل عنه : قبله ؟؟؟ خلاله ؟؟؟ بعده ؟؟؟

2. Vomiting

Analysis as above (8 as usual + for any excreta) + onset - timing - content.

(8) as usual

- Onset - course - duration.
- Association.
- What ↑ and what ↓.
- Effect of treatment.
- Date of last attack.

For any excreta

- Amount.
- Content - Color- consistency.
- Odour.

onset

- Spontaneous.
- Induced → psychic.

Timing

Early morning	Late in the day	Relation to meal
Central causes	Pyloric obstruction	➤ During → esophageal. ➤ ½ hour after → G. ulcer. ➤ 3 hours after → D. ulcer.

Content

Mucous	Chronic gastritis - cancer
Bile	Intestinal obstruction
Fecal	Intestinal obstruction
Parasite	Ascaris
Stone	Fistula
Blood	See (Haematemesis)

Polyphagia with weight loss ?

- Uncontrolled diabetes Mellitus.
- Thyrotoxicosis.
- Parasitic infestation.
- Malabsorption (Steatorrhea).

Parorexia (Pika) ?

- Anklystoma.
- Pregnancy.
- Hypercalcemia.
- Hysterical.

3. Hiccough

Definition : abnormal spasmodic contractions of the diaphragm (phrenic nerve irritation).

Aetiology

General	Chest	Neck	Abdomen
➤ CRF ➤ Psychic	➤ Carcinoma ➤ Empyema ➤ Pericarditis	➤ Rapid thyroid enlargement	➤ Subphrenic abscess ➤ Hepatic abscess ➤ Splenic abscess ➤ Acute gastric distention ➤ Haemo peritoneum

4. Changes in the bowel habit

Constipation	Tenesmus	Diarrhea
Having a bowel movement fewer than 3 times per week. With constipation stools are usually hard, dry, small in size and difficult to eliminate.	Is the feeling that you need to pass stools, even though your bowels are already empty. It may involve straining, pain and cramping.	Is the condition of having 3 or more loose or liquid bowel movements per day. "passage of more than 250 gram stool per 24 hours, provided that Pt. on western diet."

Mechanisms of diarrhea ?

1. **Hyper motility:** Exaggerated gastro-colic reflex e.g. D.M. - thyrotoxicosis.
2. **Osmotic:** Related to food e.g. lactulase.
3. **Secretory:** Infection - U.C.
4. **Spurious:** after prolonged constipation due to fermentation - rectal masses.
5. **Steatorrhea.**

Dysentery: Diarrhea + tenesmus

(passage of mucus & blood with stool) هل فيه تَقَل أو تعنية مع البراز ؟؟؟

Causes

1. **Infective:**
 - Bacterial: bacillary.
 - Parasitic: amoebic and B.
2. **Metabolic:** C.R.F.
3. **Toxic:** Mercury poison.
4. **Local causes:** diverticulosis, ulcerative colitis "U.C.", cancer colon or rectum.

Melena: Passage of soft black tarry offensive stools, due to upper GIT bleeding (from the oropharynx to end of mid gut) هل هناك براز أسود زي الزيت ؟؟؟؟

- **Site:** above ileo-cecal junction.
- **Amount:** not less than 60 ml.
- **Duration:** > 8 hours.
 - If less than 60 ml, it is called occult blood in the stool.
 - If the level below the ileo-cecal junction or the duration less than 8 hours, it is called bleeding per rectum.
- **D.D. with iron therapy or Bismuth:**
 - History of drug intake & constipation.
 - Hard stool with non offensive odour.

2. General symptoms

- **FAHM**: in L.C.F., hepatic and Hepatocellular carcinoma.

دي بتسأل فيها عن ال fever وال headache وال anorexia وال Malaise

3. Gynecological symptoms

دي بتسأل عليها في حالات ال females فقط

1. Bleeding.
2. Pain.
3. Discharge.
4. Infertility.
5. Mass (abdominal or mass protruded from the vulva).
6. Urinary (frequency, incontinence and dysuria).
7. Cessation of menstruation.
8. Hirsutism.
9. Hot flushes.

4. Pain

Don't forget (11 points)

- 8 as usual + 3 (site, radiation and character).

Medical causes of Abdominal Pain ?

- Pneumonia - angina - pleurisy .
- Hepatitis - hepatic congestion - hunger pain - gastritis.
- D.K. A.- F. M. F. - pancreatitis - Nephrotic crisis.
- Tabes dorsalis - H. Z. - Haemolytic crisis - dysmenorrheal.

Recurrent abdominal pain ?

- P.U. - Peritonitis - Pancreatitis - chronic diverticulosis - cancer colon - chronic I. O.

Types of splenic pain

Stretching of the capsule	Stretching of ligament	Splenic infarction	Splenic abscess
Dull aching	Dragging	Perisplenitis Stitching stabbing	Splenic abscess

5. Biliary (Hepatobiliary)

Symptoms:

- Jaundice.
- Pruritis.
- Dyspepsia.
- Pain.

Jaundice

Definition: yellow discoloration of skin & mucous membranes due to increase level of bilirubin > **2.5 mg %**.



لون عينيك بقى أصفر؟؟ ولون البول والبراز اتغير؟؟؟

Analysis of the jaundice**a. Onset:**

- Acute: viral hepatitis, calcular obstruction.
- Gradual: malignant obstruction, cirrhosis.

b. Course:

- Progressive: malignant obstruction, cirrhosis.
- Regressive: viral hepatitis.
- Intermittent: calcular obstructive jaundice, periampullary carcinoma, hemolytic jaundice, chronic active hepatitis.

c. Duration:

- Short: viral hepatitis.
- Long: cirrhosis.
- More than 2 years exclude malignancy.

d. Urine:

- Dark: hepatocellular & obstructive.
- Pale: hemolytic.

e. Stool:

- Pale clay: in obstructive.
- Dark: in hemolytic.
- Slightly pale in hepatocellular.

f. Anorexia, nausea, vomiting:

- Occur at the onset of viral hepatitis.

g. Fever:

- Hepatocellular: pre-ectric phase of viral hepatitis.
- Hemolytic: during hemolytic crisis - malaria - incompatible blood transfusion.
- Obstructive: Charcoat's triad.

h. Bleeding tendency: from skin, orifices in:

- Liver cell failure.
- Obstructive jaundice.

i. Pain:

- Hepatocellular: dull-aching pain in right hypochondrium in case of viral hepatitis.
- Hemolytic: bone pain and abdominal pain in hemolytic crisis.
- Obstructive:
 - ✓ Biliary colic (calcular obstruction).
 - ✓ Epigastric pain radiating to the back (malignant obstruction).

j. Pruritis:

- In obstructive jaundice.
- Primary biliary colic.

- Viral → acute - short - regressive.
- Calcular → acute intermittent .
- Malignancy → gradual - progressive (2 years).
- Chronic liver disease → gradual - intermittent (2 years).

إحنا عندنا أنواع ال " jaundice أو الصفرا "
تلت أنواع رئيسية

- Hemolytic
- Obstructive
- Hepatocellular

وأحسن ناس تشرح ال hemolytic jaundice الأطفال
إسمعنا؟؟؟

عشان الأمراض اللي بتسببها congenital ،، فبتيجي في الأطفال ،، عشان كده يا أولاد طول ما إحنا في الأطفال دماغنا في ال jaundice تروح على ال hemolytic

في الباطنة ،، الكبار ،، نركز على ال hepatitis وال hepatocellular

تروح الجراحة ،،، تنقطع في ال obstructive
فاهمين الفكرة إزاي 😊

لو خدنا فكرة مبسطة عن ال metabolism بتاع ال bilirubin
وجاي منين ،،

ببساطة خالص :

إحنا عندنا ، نوعين من ال bilirubin :

١. نوع أدام حضراتكم ، مكتوب باللون الأحمر ، ال Indirect bilirubin

ال Indirect bilirubin هعرفه إزاي clinically ؟؟؟

أعرف إزاي إن العيان الي أدامي ده عنده ال Indirect bilirubin هو الي عالي ؟؟؟

أول حاجة ، لون العيان هيقا شكله إيه ؟؟؟

هتلاقي الصفار بتاع العيان ، لونه golden yellow ، أصفر ذهبي

يعني الصفار بتاعه بيميل إلى اللون الذهبي شوية " وده غالباً في الأطفال يعني "

طيب ،

افترضنا فيه ناس كثيرين ، معندهمش القدرة على تمييز الألوان ،

يعرف إزاي ؟؟

عندنا ال Indirect bilirubin

fat soluble

أدام fat soluble ، عمره ما ينزل في ال urine ، وبالتالي عمره ما يغير من لون ال Urine

عشان هو إيه ؟؟؟ fat soluble

ولذلك ،

تلاقي الواد عينيه صفرا ، ولون البول طبيعي ،

Normal colored urine

طيب ،

أومال هينزل عن طريق مين ؟؟؟ ال biliary system

لما ينزل عن طريق ال biliary system

النتيجة هينزل هنا ، في ال stool زي ما هنتكلم على ال Metabolism بالتفصيل إن شاء الله

هيزود كمية ال stercobilinogen وال stercobilin في ال stool

يخلي ال stool ده إيه ؟؟؟ لونه dark in color

لكن ،

أي عيل هتلاقيه لون أصفر ، أسأل الأم ، ال urine أخباره إيه ؟؟؟

إذا قالتلك : No change in the color of urine ،

يبقا ده ال Indirect hyper bilirubinemia

ملكش دعوة بلون إيه ؟؟ ال stool

يبقا الولد إذا كانت عينيه صفرا ، ومعاه normal colored urine

يبقا ده ال Indirect hyperbilirubinemia

٢. النوع الثاني من ال bilirubin الي هو اللون الأزرق ده الي هو ال direct bilirubin

ال direct bilirubin ، يتوصل بال blood هيخلي الولد لونه أصفر

بس الصفار بتاعه بيميل للخضار شوية

عامل زي لون الليمون ، Lemon jaundice

لأن الليمون مصفر بس بيدي على الخضار شوية

يبقا هنا اللون أصفر ، بيرمي شوية على greenish color

ال direct bilirubin ،

ده water soluble

وأدام water soluble ، هينزل فين ؟؟؟ في ال urine

ال bilirubin لو نزل في ال Urine هيخلي لون ال urine غامق ،، هيبقا لونه إيه؟؟؟
عامل زي الشاي الفاتح أو الكوكاكولا
يبقا هنا يعمل tea colored urine

يبقا هنا الأمور منتهى البساطة ،، الواد مصفر؟؟ أبوة
أسأل الأم البول أخباره إيه؟؟؟
متغيرش؟؟؟ يبقا Indirect hyperbilirubinemia
تغير؟؟؟ يبقا ده direct hyperbilirubinemia

طيب ،،
هنبداً نراجع مع حضراتكم سريعاً الكلام
metabolism of bilirubin ال

كلكم عارفين هذا الكلام ،،
إحنا عندنا ال indirect bilirubin بيطلع من ال RBCs
Once إن ال RBCs حصلها hemolysis
نتيجة ال Metabolism of hemolysed RBCs
هيبداً يطلع عندنا ال Indirect bilirubin

Indirect bilirubin ده ،، very toxic
معنى ،، إن هو fat soluble ،،، يستطيع أنه يعدي ال blood brain barrier
ولو وصل إلى ال brain stem هيدمرها
هيعمل حاجة اسمها bilirubin encephalopathy

يبقا هو very toxic to the brain stem
ويستطيع أن يصل إلى ال brain stem عشان هو fat soluble

ربنا سبحانه وتعالى ،، عملنا حماية قوية جداً من ال indirect bilirubin
إزاي؟؟؟

إن ال Indirect bilirubin ده بشيله مين؟؟؟
albumin ال

albumin ال هو الي بشيل ال Indirect bilirubin
albumin ال ،،
molecular weight بتاعه لا يسمح له إن هو يعدي ال blood brain barrier
albumin ال ،، مبعديش ال blood brain barrier (طول ما ال blood brain barrier سليم)
وبالتالي ما يحمله ال albumin لا يستطيع إن هو يدخل جوا ال blood brain barrier
يبقا هنا ال Indirect bilirubin ،،

عمره ما هيقدر يعدي ال blood brain barrier بالرغم إن هو fat soluble
ليه؟؟؟
لأنه ماسك في ال serum albumin

طيب ،،
أومال إمتى ال Indirect bilirubin يعدي ال blood brain barrier؟؟؟
لو أصبح free bilirubin ،،، إن هو مش ماسك في ال serum albumin

طبيب «

الكلام ده هيجصل إمتي ???

وجدوا الأتي :

إن عندنا كل واحد جرام من ال albumin بشيل من 6 إلى 8 ملي جرام من ال indirect bilirubin
ييقا كل واحد جرام من ال albumin بشيل من ستة إلى ثمانية ملي جرام من ال indirect bilirubin

طبيب «

لو واحد عنده say «

ال serum albumin بتاعه 4 جرام « أقصى كمية bilirubin يشيلها أد إيه ???
إحنا بنحسب على ال Low «، الي هو كام ??? 6 ملي جرام

ييقا هنا لو واحد عنده ال serum albumin « أربعة
يقدر يشيل أد إيه من ال indirect bilirubin ??? يشيل 24 ملي جرام من ال Indirect bilirubin

الأربعة وعشرين دي جت إزاي ???

إن أنا عندي الجرام ألبومين يشيل ستة ملي جرام من ال Indirect bilirubin
ييقا هنا عندي 4 جرام أقدر أشيل كام ??? أقصى كمية من ال indirect bilirubin أشيلها أربعة وعشرين ملي جرام

لو زاد عن كده ال indirect bilirubin

الألبومين ميقدرش يشيل

ليه ???

لأن ال albumin وصل لل saturation level

يصبح ال Indirect bilirubin عندي free

وأدام أصبح ال indirect bilirubin عندي free «

لأن ميقاش ليه أماكن في ال albumin يمسك فيها « في الحالة دي يستطيع ال Indirect bilirubin يعدي ال blood brain barrier
ولو عدى ال blood brain barrier هيعملنا bilirubin encephalitis

ييقا هنا « أنا طول ما ال bilirubin معداش ال saturation level بتاع ال Albumin

أنا مش خيف من موضوع مين ??? ال kernicterus

طبيب « ربنا سبحانه وتعالى بقا « إزاي يخلصني من ال Indirect bilirubin ???

ال Indirect bilirubin « زيه زي أي حاجة fat soluble

(الناس الي سرحانه)

ال main site of excretion مين ??? ال Liver cells

يقوم رايح على ال Liver cells

ال Liver cells « تبدأ تعمله uptake من البوابة دي

عن طريق عملية إسمها endocytosis

يعني بتبدأ تدخله في صورة vesicle « وتدخله جوا مين ??? الخلية

عملية ال Uptake دي « مين الي بيتحكم فيها ???

بيتحكم فيها نوعين من البروتين جوا ال Liver cell

• نوع إسمه Z protein

• نوع إسمه Y protein

هذه الأنواع من البروتينات مش موجودة في ال blood « موجودة في ال liver cells

طيب ،

ما وظيفة ال Z و ال Y protein ؟؟؟

قالوا : إن ال Indirect bilirubin يا أولاد لو دخل جوا ال Liver cells
وأصبح هو نفسه جوا ال Liver cell ، free ، ، ،
هيدمر ال Liver cells

لذلك ، ، ربنا سبحانه وتعالى ،

جاعل ال Z وال Y protein يمسكوا في ال Indirect bilirubin
يمنعوا ال Indirect bilirubin إنه يتسبب ويدمر ال Intra cellular component بتاعت ال liver cell
اللي هي الميتوكوندريا وال endoplasmic reticulum وال ribosomes والحاجات اللي جوا دي كلها
طول ما ال Indirect bilirubin ماسك في ال Z وال Y protein

يبقا هنا ،

وظيفة ال Z وال Y protein ،

هي نفس الوظيفة اللي بيعملها مين ؟؟ ال albumin

ال albumin بيمسك في ال Indirect bilirubin يمنع ال bilirubin يدخل جوا ال brain cells ويدمرها
ال Z وال Y proteins ، بيمسك في ال Indirect bilirubin جوا ال Liver cells يمنع إن ال Indirect bilirubin إن هو يدمر ال Intra cellular
component بتاع مين ؟؟ ال Liver cells

طيب ،

لذلك ، ، عندنا ال Z وال Y proteins

قولنا هما اللي بيتحكموا في دخول ال Indirect bilirubin

ليه ؟؟

Once إن ال Z وال Y protein بقوا saturated

ال Uptake يقف ،

عارفين ليه ؟؟؟

لأن أدام هما saturated مش هيقدرنا يشيلوا أكثر من كده ،

أي Indirect bilirubin يدخل هيصبح إيه ؟؟؟ free

ولو أصبح ال Indirect bilirubin free ، هيدمر ال Liver cells

يبقا اللي بيتحكم في ال uptake بتاع ال Liver cells

هما ال Z وال Y proteins

Once إن هما saturated ، ال Uptake يقف

طيب ،

ما هو ربنا عايز الأمور تستمر ،

يقوم ربنا يعمل إيه ؟؟؟ مخلي ال Z وال Y protein ، لهم القدرة إنهم يتخلصوا من البلوة اللي هما شايلينها

اللي هو ال Indirect bilirubin

يعملوا إيه ؟؟؟

يبدأوا يخلوا ال Indirect bilirubin ، يتفاعل مع حاجة جوا ال Liver cells اسمها glucouronic acid

عن طريق إنزيم اسمه glucoronyl transferase enzyme

يبعمل حاجة إسمها إيه ؟؟؟ conjugation

يمسك ال glucouronic acid في ال indirect bilirubin

ويكون لي حاجة اسمها bilirubin glucuronyl أو glucouronic acid bilirubin
اللي إحنا بنقول عليه direct bilirubin

direct bilirubin ال

Is non toxic « مفيش منه أي مشكلة

مش كده ويس «

direct bilirubin ده « water soluble « بيقا هنا ال glucururonic acid once إنه مسك في ال Indirect bilirubin

• هحوله من toxic إلى Non toxic

• حوله من fat soluble إلى water soluble

غير من الخصائص بتاعته تماماً «

طيب « ال direct bilirubin نخلص منه إزاي ???

Liver cells « تبدأ ترميه جوا ال intra hepatic fine canaliculi

اللي هي موجودة في كل ال Liver

Liver cells ال هنتمي ال direct bilirubin « في هذه ال intra hepatic fine canaliculi

عن طريق active excretion

بيقا ال direct bilirubin يطلع من ال Liver cells

ويتمى جوا ال intra hepatic fine canaliculi عن طريق active process

ATP consuming mechanism

direct bilirubin ال

زي ما كلنا عارفين « Once إن هو excreted في ال intra hepatic fine canaliculi

هيمشي مع ال bile secretion

لما يمشي مع ال bile secretion يوصل لل gut

يلاقى عندنا مين ??? ال bacterial flora

ال bacterial flora « هتعمل إيه ???

هتحوله إلى stercobilinogen

ال stercobilinogen ده «

90 % منه هينزل في ال stool ويحصله oxidation ويتحول إلى ال stercobilin

اللي بيخلي ال color بتاع ال stool لونه بني شوية

مش كده ويس «

فيه 10 % هيبقوا absorbed عن طريق عن ال Portal circulation ويرجعوا مرة ثانية لمين ??

لل Liver

ال 10 % بتوع ال stercobilinogen «

اللي راجعين في ال enterohepatic ما وظيفتهم ???

وظيفة مهمة جداً

إن هي عبارة عن negative feedback mechanism

بيعمل إيه ??? يعمل ال suppression ال glucuronyl transferase enzyme

بيقا هنا وظيفة « ال stercobilinogen الي راجع في ال enterohepatic إنه يوقف شغل ال glucuronyl transferase enzyme

فيقلل كمية مبن ؟؟ ال direct bilirubin
فيقلل كمية ال stercobilinogen

عشان ،

لو ال stercobilinogen ، زاد ، أو ال direct bilirubin ينزل في ال bilirubin
هيعمل مشاكل ، هنعرفها في حينها إن شاء الله

يبقا هنا عمل inhibition لل enzyme عشان ينظم كمية ال direct bilirubin
متزددش عن قدر معين
إنه يعمل suppression لل glucoronyl transferase enzyme

وفيه عندنا ،

جزء منهم بيوصل للدم ، وينزل في ال urine في صورة إيه ؟؟؟ urobilinogen
ده ال Metabolism of bilirubin

بعد كده نتكلم على أنواع ال jaundice

النوع الأول Hemolytic jaundice

جاية نتيجة hemolysis

ال RBCs تطلعلي hemoglobin بالزيادة

معنى كده unconjugated bilirubin زادت هيفضل يلف في الدم مينزلش في ال Kidney عشان حجمه كبير ومبيذوبش في الماء

يروح على ال Liver هيعمله uptake

بس خلي بالك ال unconjugated bilirubin عالي وال uptake بردو عالي

هيعمل conjugated bilirubin جوا ال Liver كثير ، والسكة سالكة ، ينزل في ال intestine يقوم يزود ال stercobilinogen

يقوم يزود ال stercobilinogen يقوم يزود بردو urobilinogen كل حاجة هنا زيادة

- ال bilirubin هنا عالي
- ال stercobilinogen عالي
- ال stercobilinogen عالي
- ال urobilinogen عالي

بس خلي بالك هنا من حاجة مهمة ال hemolytic jaundice مهما كانت severe

أي مهما كان ال hemolysis كبير فلا يمكن ال bilirubin أن يعلى عن خمسة عشان كده يقولك ال hemolytic jaundice مبتصفرش أوووي بتوع الأطفال عشان دماغهم صغيرة يقوموا يقولوك بيكون لونهم أصفر ليموني ☺

فلما يجيلك واحد وتلاقي ال bilirubin بتاعه سبعة أو تسعة معنى ذلك ان ده مش hemolysis

وكمان ال Liver تعبان أما لو سليم لا يمكن أن يعلى ال bilirubin ، أن يعلى عن خمسة

لون ال urine هنا مش هيتغير على الرغم ان ال urobilinogen كثير بس هو أصلا ملوش لون

لون ال stool هنا هيكون ؟؟؟؟ غامق عشان فيه stercobilinogen كثير هيتحول الى stercobilin وال stercobilin بيغمق ال stool

نجيب من الآخر أكثر حاجة بتصفر في ال jaundice هي لون ال Urine ولون ال stool

سؤال بإجابته ؟☺

س/ إيه هي نوع ال Jaundice اللي فيها لون ال urine مش غامق ؟؟؟؟

ج/ الاجابة هي hemolytic jaundice بس خلي بالك ال stool بيكون غامق

مينفعش عيان يجيلك لون ال Urine وكمان ال stool كويس أو مال إيه اللي جابه ؟؟؟

العكس بقا في Hepato-cellular jaundice وال Obstructive jaundice

obstructive jaundice النوع الثاني

من الاسم واضحة فيه obstruction طيب فين ال Obstructive لكن ال unconjugated نسبته normal conjugated bilirubin يبقى ال conjugated bilirubin هو اللي بيعلي في ال Obstructive لكن ال unconjugated نسبته normal ولكن هذا ال conjugated bilirubin حجمه صغير ويذوب في الماء يبقى هينزل في ال urine وطالما يينزل في ال urine ومعروف إن ال conjugated bilirubin لونه غامق هتلاقي ال Urine لونه غامق وفي نفس الوقت هما انه فيه obstructive والطريق مسدود هيكون نسبة ال stercobilinogen في ال Intestine قليلة بالتبعية هيكون ال stercobilin هيكون قليل وجايز دا اللي بيخلي لون ال stool فاتح أووووي بس خلي بالك ال urobilinogen قليل ما هو هو ال stercobilinogen صح ولا غلط يبقى نستخلص من كده في ال obstructive jaundice ال conjugated bilirubin بتعلي وبيخلي لون ال urine غامق ال stercobilinogen وال urobilinogen قليل

بس خلي بالك هو اللي يينزل في ال bile هو ال bilirubin بس؟؟؟؟ ولا حاجات تانية طبعا حاجات تانية زي ال bile salts اللي هي كمان عشان الطريق مسدود مش هتتعرف تنزل هي كمان تقوم تعلى في الدم فيقولك جايز ال bile salts قد تكون هي سبب ال itching وال bradycardia اللي بتكون في العيان ده بس الكلام ده مش مضبوط ده أووووي جايز اللي بيعمل itching حاجات زي ال prostaglandins اكتشفوها حديثا وجايز تقوله في ال Itching الكلام الحديث unknown

خلي بالك معنى إن ال bile موصلتش ال intestine يبقى مش هتتعرف تمتص ال vitamins بتاعت أكيد AKED وخلي بالك كمان اللي يينزل كمان ال cholesterol أظن دي الحالة الوحيدة اللي في ال jaundice اللي تعلي ليك ال cholesterol صح ولا غلط؟؟؟ صح وكمان خلي بالك اللي يينزل كمان ال alkaline phosphatase مش هيعرف ينزل هيزيد في الدم

😊😊😊😊😊😊😊😊

خلي بالك ال bile salts مش عارف ينزل يرجع لل Liver يجيبك biliary cirrhosis عشان كده يقولك الجملة المشهورة Long standing obstructive jaundice بتجيب liver cirrhosis

النوع الثالث Hepatocellular jaundice

يجيب من الآخر اكتب كلمة واحدة أنها مرحلة وسط ما بين ال conjugated bilirubin و ال unconjugated bilirubin يزيدوا
 عندنا ال Liver تعبان معرفش يعمل uptake ماش كويس بيقا مش كل ال Unconjugated هتعرف تدخل بيقا هتزيد في الدم بيقا اول كلمة
 ان ال Unconjugated bilirubin بتكون عالية عشان ال Uptake ماش كويس
 المهم شوية Unconjugated يدخلوا ال Liver يتعملهم conjugation بعد ما يتعمل conjugation في ال liver
 ياخذ بعضه وينزل عشان يتعمله excretion
هل ال excretion كويس ؟؟؟؟ طبعا لا عشان ال hepatocyte تعبانة
 وطالما ال excretion ماش كويس ال conjugated bilirubin ترجع للدم وتعل فيهِ

بالمُناسبة إيه اللي بيعلى أكثر ال conjugated ولا ال Unconjugated؟؟؟؟☺
 ج/ اللي بيعلي أكثر ال conjugated
 عشان داهما لما ال liver يوظ أكثر عملية تتأثر هي ال excretion وليس ال uptake

المهم ال conjugated bilirubin معرفش ينزل مع ال bile ويوصل لل intestine بيقا ال stercobilinogen قليل بيقا ال stercobilin قليل بيقا
 بردو ال urobilinogen قليل؟؟؟
 غلط xxxxxxxxxxxxxx بيكون زايد دا الصح
 على الرغم في الأول قولنا ان ال urobilinogen هو هو ال stercobilinogen
 السبب اللي خلي urobilinogen يزيد
 obstructive ال Hemolytic ال Hepatocellular jaundice مرحلة وسطى بين ال ال obstructive

- في ال hemolytic كان ال Urobilinogen وال stercobilinogen الاتنين بيعلوا
- وفي ال obstructive الاتنين بيعلوا
- في ال Hepatocellular أنا بقولك أن ال stercobilinogen بيقل معنى كده أن ال urobilinogen بيزيد عشان واحدة تزيد واحدة تقل عشان يكون مرحلة وسط
- السبب المقنع بقا سيبك انت من التحشيش ده
- ال stercobilinogen لما يوصل ال intestine ادامه التلت خيارات ☺☺☺
- الخيار الاول ان التلت ينزل في ال stool على هيئة stercobilin
- والخيار الثاني Entero hepatic circulation
- والخيار الثالث يروح لل kidney عشان ينزل على هيئة urobilinogen
- في Hepatocellular jaundice تلت ال stercobilinogen ينزل في ال stool بعد ما يتحول الى stercobilin والتلت الثاني يجي يروح لل Liver
- فال Liver يقوله إيه ؟؟؟ يا بني أنت أهبل أنت بتعمل إيه أنت مش شايف اني منزلك بالعافية دا فيه Hepatocellular jaundice دا ال excretion طلع عنيا عشان اعرف انزلك تقوم ترجعلي ثاني هيصبح المصير بتاع التلت ده ينضم للجزء الي يروح للكلية مع ال urobilinogen
- بيقا التلت يروح على هيئة stercobilin والتلتين يروحوا على هيئة urobilinogen

6. Uro- genital system البول

- طبعاً ال urogenital system ده يعتبر system ثاني ،
- بس أنا مدخله ضمناً معانا
- عشان يهمني ال pain during micturation ، إذا كان عنده dysuria ، إذا كان عنده frequency ،
- إذا كان عنده percipitancy
- ومشاكل ال genital system ، إذا كان عنده impotence أو حاجة لازم بردو أسأل عنها

1. Micturation

- Pain.
- Micturation difficulties (dysuria).
- Urine abnormalities: volume - colour - frothy.
- Uraemic symptoms.

2. Sexual

- Desire.
- Erection troubles.

7. Abdominal Swelling

تسأله بطنك ورمت ولا انتفخت ولا لا ؟؟

Localized	Generalized
➤ Spleen. ➤ Liver. ➤ Mass.	Ascites

History of aspiration

1. Amount.
2. Aspect.
3. Colour (red = hge.).
4. Complications: fever (infection), wound sepsis, shock & hepatic encephalopathy.

8. Lower limb Swelling

رجلك ورمت ولا لا ؟؟؟؟

- L.C.F.
- Bilharziasis.
- Cor-pulmonale.

9. Systemic review

iii. Other systems

متنشاخ الخطوة الثالثة Other systems

هتسأل بقا سؤاين في ال cardio على ال Pulmonary congestion وال systemic congestion
 وال manifestation بتاعت ال low cardiac output وال syncope
 الحاجات الكبيرة دي بتسألها على السريع
 بالنسبة لل neurology بتسأل عن الحاجات الكبيرة
 motor weakness, sensory affection يعني مش بالتفصيل الممل
 ولو مفيش فيها أي حاجة
 no symptoms suggest other systems affection
 وتكتبها كده يبقا انت سألت عليها
 عشان محدش يجي يقولك أنت مسألتهش على ال Other systems ليه !!!!

iv. Investigation & treatment (related diseases)

اللي إنتوا بتضيعوا فيها بدون مبالغة عشر دقائق
 وهي في الآخر تسوي إيه ؟؟؟ تسوي ولا حاجة

لو نستها ممكن تعدي
 إحنا مش هننساها إن شاء الله ومش هنضيع فيها وقت
 متقعدش تقولي العيان راح فين وجه منين ؟؟؟
 تقولي :

Patient sought medical advice
 admitted فين ؟؟؟

و recommended إيه ؟؟؟
 وعملوا إيه ؟؟؟

وخرج من المستشفى
 واتحجز في المستشفى الفلاني
 وبعدين اتحجز في الثاني وبعدين مش عارف
 إحنا ملنا بالكلام ده !!!!!!!

أنا عايز جملة واحدة
 patient investigated by ECG, chest X-ray, ECHO cardiography
 والدوبلر

and treated by Beta blocker
 خلاص أنا مش عايز حاجة ثانية
 أنا عايزها كده جملة في الآخر
 متقعدش تفتح في حكاوي العيان ، لازم تلجم كده العيان في الامتحان
 وإلا مش هنخلص يعني ماشي ☺

SUMMARY OF HISTORY OF PRESENT ILLNESS IN ABDOMEN

G.I.T. (upper)

- شهيتك على الأكل اتغيرت؟؟ هل هناك صعوبة في البلع للسوائل أو للأكل؟؟
- حصلك ترجيع؟؟ رجعت دم قبل كده؟؟
- بتحس باللغاب مالي فمك؟؟ ونفسك غامة عليك؟؟
- رائحة فمك متغيرة؟؟ هل عندك حمو على فم المعدة؟؟
- بتتكرع؟؟ بجيلك زغطة؟؟

G.I.T. (lower)

- بطنك انتفخت؟؟ وفيه غازات؟؟ Flatulence
- فيه دم أحمر مع التبرز؟؟ haematochazia
- هل هناك براز أسود زي الزيت؟؟ melena
- فيه إسهال أو تعنية؟؟ فيه أصوات من البطن؟؟

General

- فيه ارتفاع في درجة الحرارة؟؟ فيه صداع؟؟
- فيه فقد للشهية؟؟
- فيه توعك وهمدان في جسمك؟؟

Gyneacological

- فيه ألم؟؟ فيه نزيف؟؟ فيه إفرازات؟؟

Pain

- عندك ألم في بطنك؟؟ بيزيد بإيه؟؟ وبيققل بإيه؟؟ ومتنساش ال 11 نقطة

Biliary system

- لون عينيك بقى أصفر؟؟ ولون البول والبراز اتغير؟؟

Urinary and genital system مشاكل في البول

- فيه ألم عند التبول؟؟ البول بيسبقك؟؟
- عندك ضعف جنسي؟؟

Abdominal Swelling

- بطنك ورمت؟؟ أو انتفخت ولا لا؟؟

Lower limb Swelling

- رجلك ورمت أو تفتحت ولا لا؟؟

Systemic review

- فيه حاجة تانية بتشتكي منها في بطنك؟؟
وتسأل المريض بردو
وتقوله :
هل عندك مشاكل في أي جزء ثاني في جسمك؟؟
عملت فحوصات إيه؟؟
وأخذت علاج إيه؟؟

Past history

Diseases	Operations	Drugs
➤ Bilharziasis. ➤ Hepatitis. ➤ T.B. ➤ D.M. ➤ Hypertension.	➤ History or blood transfusion.	Hepato-toxic A. Dose dependant • Paracetamol 16 gm. B. Non dose dependant • Acute necrosis → INH - PAS. • Cholestasis → aldomet. • Tumors → C. pills

Bilharziasis	hepatitis	
➤ History of contact to canals. ➤ Clinical pictures of Bilharziasis. ➤ Investigations	Subclinical	Clinical
	Unnoticed	➤ Fever. ➤ Jaundice. ➤ Dark urine. ➤ Fever hospital.
Easily excluded لو حد عمره ما نزل التربة أو شرب منها		Can't excluded

N.B.

- Bilharziasis never causes L.C.F.

Examination

General

As usual + functional evaluation

A أرقام		➤ Respiratory distress: tense ascites - pleural effusion. ➤ Hypotension: shocked - postural hypotension (↑ VDMs). ➤ Pulse: • Tachycardia: hyperdynamic circulation (↑ VDMs). • Bradycardia: in obstructive jaundice. ➤ Fever → low grade.
Built		➤ Underbuilt: weight loss and wasting (L.C.F. - carcinoma). ➤ Overbuilt: ascites (oedema) - autoimmune treated by cortisone. ➤ Dwarfism: liver disease since childhood.
Colors		➤ Pallor: Anaemic (all types may occur). ➤ Jaundice (types). ➤ Hyperpigmented: (Hemochromatosis - L.C.F.). ➤ Cyanosis: tense ascites - porto-pulmonary - A.V. shunt.
Decubitus		➤ Orthopnea: tense ascites - pleural effusion.
E	فوق	➤ Congested neck veins: tense ascites - pleural or pericardial effusion. ➤ Head and neck (see below).
	منتصف	➤ Clubbing • Crohn's - Ulcerative colitis. • Biliary cirrhosis. • B. polyposis. • Intestinal steatorrhea. ➤ Flapping tremors.
	تحت	➤ Lower limb edema. ➤ Limbic ex (see below).
F	فكر	➤ Disturbed mentality (Hepatic encephalopathy).
	Facial	➤ Temporalis muscle wasting + enlarged parotid.

Enlarged lymph node due to:

- Supraclavicular nodes (Virchow's node): lung or GIT malignancy.

Functional evaluation

A. Vascular decompensation (portal hypertension)

Evidences of portal hypertension:

1. **Splenomegaly:** the single most important diagnostic sign. And may lead to hypersplenism.
2. **Ascites:**
 - Early ascites: acute portal or splenic vein thrombosis.
 - Late: related to additional factors as hypo-albuminaemia.
3. **Portosystemic anastomosis:**

	Location	Portal circulation	Systemic circulation	Consequences
1.	Gastro-esophageal	Coronary V. of stomach	Azygos vein	Gastro - esophageal varices
2.	Anorectal	Middle and superior hemorrhoidal V.	Inferior hemorrhoidal vein	May be mistaken for hemorrhoids
3.	Anterior abdominal wall	Para-umbilical V. in falciform lig. (left portal vein)	Superior & inferior epigastric V.	Caput medusae
4.	Retroperitoneal	• Splenic V. branch. • Veins around liver and diaphragm. • Visceral veins.	• Left renal V. • Abdominal wall V. • Retroperitoneal V.	Between: 1. Liver & diaph. 2. Spleen & diaph. 3. Intestine & post abd. Wall.

Presentation of Portosystemic anastomosis:

1. Asymptomatic (diagnosed by ultrasound or endoscopy).
2. Haematemesis.
3. Internal bleeding.
4. Chronic encephalopathy.

B. Parenchymatous decompensation (L.C.F.)

Manifestations of liver cell failure

- **F**ever (low grade).
- **F**etor hepaticus D.D.
- **F**lapping tremors.
- **F**atigue.
- Jaundice D.D.
- Encephalopathy.
- Ascites.
- Skin changes:
 - Spider naevi D.D. - palmar erythema - white nails (↓ protein).

- Endocrinal
 - Gynecomastia.
 - Testicular atrophy.
 - Feminine pubic hair.
 - Hyperaldosteronism.
- Blood
 - Anaemia (pallor).
 - Bleeding tendency.
- C.V.S.
 - Hyperdynamic circulation.
- Kidney
 - Hepato-renal failure.

Spider naevi

- Central dilated arteriole with radiating capillaries (looks like a spider).
- Pressure on central arteriole leads to fading.
- Releasing of pressure: the blood refill the “legs of the spider”.
- ***Distributed in the upper half of the body (course of the S.V.C.).***
- ***Confirmed by:***
 1. Central pressure = blanching.
 2. Slide test = pulsation.



*One or two spider naevi may occur in normal people, thyrotoxicosis or pregnancy.
More than 5 considered as significant and likely to due to L.C.F.*

D.D.

	Level	Pruritus	Pressure	Site
Spider naevi	Raised	Not	Fading	SVC course
Insect bite	Raised	Itchy	No	Exposed area
Purpura	Not	Not	Not	LL (common)
Campbell de Morgan spots	Raised	Not	Not	Trunk
Venous star	variable	±	Fading	LL

Palmar erythema

- redness of thenar, hypothenar, distal ends of metacarpal bone, pulp of fingers with central pallor.
- **Causes:**
 - Pregenancy.
 - Thyrotoxicosis.
 - L.C.F.
 - Estrogen containing contraceptive pills.



Additional items of general examination

1. Limbic examination.
2. Head and neck examination.
3. Sexual chr. Evaluation.
4. Systemic review (C.N.S. - Chest - C.V.S. - Kidney).

1. Limbic examination.

A. Upper limb	
Nails	➤ Koilonychia (Fe deficiency due to GIT bleeding). ➤ Leuconychia (hypoalbuminaemia). ➤ Muehrke's lines (hypoalbuminaemia). ➤ Blue lunlae (Wilson's).
Hands	➤ Asterixis: patient stretches out hands in policeman's stop position and fingers spread out (+ closed eye). ➤ Palmer erythema. ➤ Pallor of the creases. ➤ Dupuytren's contracture: (fibrosis of the palm's fascia) (alcoholism, manual labor). ➤ Palmer or tendon xanthomata.
Arms	➤ Scratch marks. ➤ Spider naevi. ➤ Bruising. ➤ Xanthomata.
B. Lower limb	
➤ Oedema. ➤ Bruising. ➤ Neurological examination. ➤ Xanthomata. ➤ Toe nails and foot: showing the same symptoms as finger nails.	

2. Head and neck : fine silky hair

Eyes	➤ Cornea rings (Wilson's). ➤ Sclera: jaundice. ➤ Iritis: inflammatory bowel disease. ➤ Xanthelasma.
Lips	➤ Telangiectasia. ➤ Brown freckles (Peutz-Jaegers). → 
Mouth	➤ Breath: fetor hepaticus - Ethanol (alcoholism). ➤ Ulcer: (Crohn's, coeliac) - white candida patches - cracks at mouth edges. ➤ Teeth: cavities (acid effect). ➤ Gums: hypertrophy - bleeding - Gingivitis.
Tongue	➤ Leucoplakia: smoke, spirits, sepsis, syphilis, sore teeth. ➤ Atrophic glossitis: hypovitaminosis - Plummer vinson. ➤ Macroglossia : B12 deficiency.

3. Sexual character evaluation

e.g. gynecomastia and testicular atrophy.

4. Systemic review

	Cause	Result	Association
C.N.S.	Autoimmune PN (gastro paresis diabeticorum)	Hepatic encephalopathy	Hepatolenticular "Wilson's"
Chest	Cor-pulmonale	➤ Amoebic liver abscess. ➤ Hydatid. ➤ Sympathetic effusion (Rt. PI). ➤ Hepato-pulmonary Syndrmome.	Alpha one anti trypsin deficiency
C.V.S.	➤ Cardiac cirrhosis. ➤ S.B.E. = splenomegaly.	➤ Bilharziasis. ➤ Cor-pulmonale.	
Renal		➤ Hepato-renal. ➤ Pre-renal (acute GIT bleeding). ➤ Nephrotic (extra hepatic of HCV - B mansoni).	

Local examination

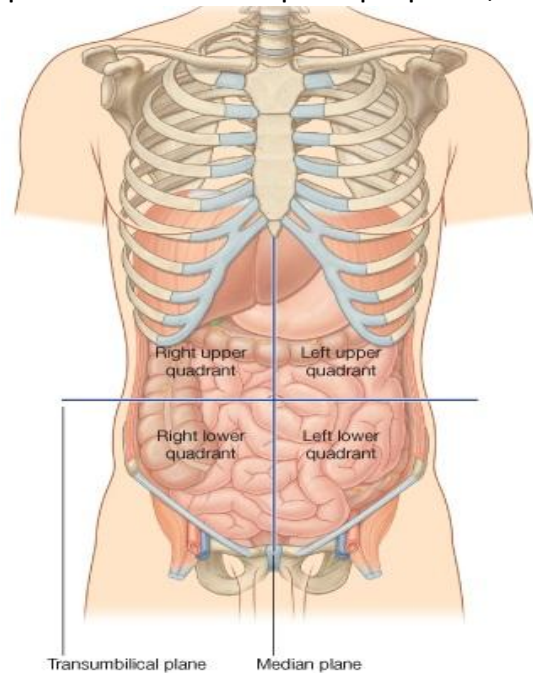
The patient is exposed from the nipple to the mid thigh and in supine position.

Abdominal quadrants

- Clinicians usually divide the abdomen into four quadrants for descriptive purposes, using the following planes:

- Median plane: imaginary vertical line following the line alba from the xiphoid process to the pubic symphysis
 - Transumbilical plane: imaginary horizontal line at the level of the umbilicus
- These lines or planes create four quadrants:

- ✓ Right upper
- ✓ Left upper
- ✓ Right lower
- ✓ Left lower

**Abdominal regions**

- Clinicians may divide the abdomen into 9 regions:
- For more accurate descriptive and diagnostic purposes
 - Use two vertical and three horizontal lines or planes

Horizontal planes (in descending order)

- Subcostal plane: passes through the lower border of the 10th costal cartilage on either side

Sometimes

the transpyloric plane is used instead of the subcostal; passes through the pylorus on the right and the tips of the ninth costal cartilage on either side)

Transumbilical plane: passes through the umbilicus at the level of the L3/4 intervertebral disc.

- Transtubercular (intertubercular) plane: passes through the tubercles of the iliac crests and the body of L5

Vertical planes

- Right midclavicular line
- Left midclavicular line

Pass from the midpoint of the clavicle to the midpoint of inguinal ligament.

These planes create 9 abdominal regions:

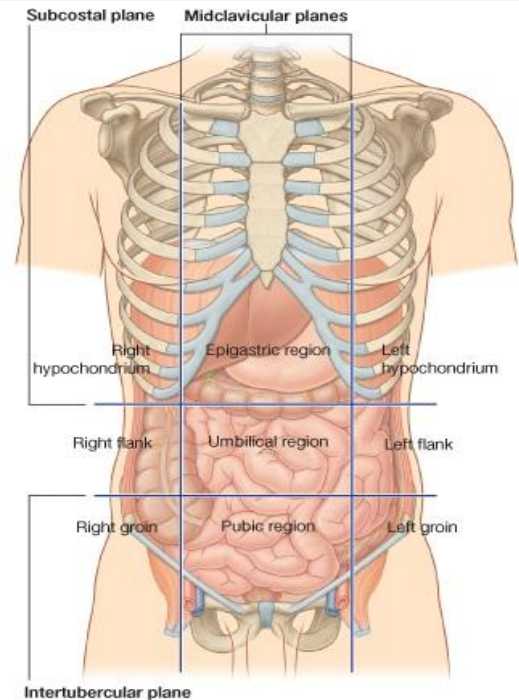
- ✓ Right and left hypochondriac regions, superiorly on either side
- ✓ Right and left lumbar (flank) regions, centrally on either side
- ✓ Right and left inguinal (groin) regions, inferiorly on either side
- ✓ Epigastric region superiorly and centrally
- ✓ Umbilical region, with the umbilicus as its center
- ✓ Hypogastric or suprapubic region, inferiorly and centrally.

Descriptive quadrants and regions are essential in clinical practice

Each area represents certain visceral structures

Allow correlation of pain and referred pain from these areas to specific organs.

Regions and quadrants are palpated, percussed, and auscultated during clinical examination



Inspection

General consideration:

- The patient must be exposed from the nipple to the symphysis pubis.
- The patient's hand should remain at his sides with head resting on a pillow.
- Flexion on the knees may relax the abdomen.
- The patient should have an empty bladder.
- Warm room and adequate light must be provided.
- For easy localization of any abnormalities it is useful to divide the abdomen in 4 quadrants or 9 segments as before.
- Watch the patient's face for signs of discomfort during the examination.

Inspection and palpation		
Front (15)		Back
Midline (7) من فوق لتحت	Sides (8)	
• Subcostal angle. • Epigastric pulsation. • Visible peristalsis. • Divercation. • Umbilicus. • Hair distribution. • Genitalia.	• Respiratory movement. • Breast. • Contour. • Hernial orifices. • Pigmentation. • Veins. • Scratching marks. • Scars.	• Scar. • Deformity. • Swelling of renal angle. • Cafe' au lait patches "neurofibroma". • Tuft of hair "spina bifida".

Inspection of the front "midline" (7)

1. Subcostal angle

- **Normal:** acute to right (70° - 90°).
- **Obtuse:** upper abdominal swelling (HSM) - ascites - barrel shaped chest.

2. Epigastric pulsation: (see cardiology) ولا خليك معانا هنا 😊

Place your hand longitudinal in the subcostal angle, Pulsation classified according to the direction into :

- At the tip of fingers: RV ++ , increased with deep inspiration.
- From the right side: hepatic pulsation (tricuspid stenosis - TR), liver is enlarged and tender during bimanual examination.
- From behind: aortic pulsation (thin - hyperdynamic - aneurysm), pulsating down to umbilicus.

3. Visible peristalsis.

- **Normal:** in thin person or in emaciated person.
- **Abnormal:**
 - a. Pyloric obstruction: slow waves from the left rib margin to the right. Exaggerated by massage, tapping or drinking soda + confirmed by Succussion splash (see auscultation).
 - b. Intestinal obstruction: called step ladder.

4. Divercation of recti : Separation of rectus abdominis muscles

- Ask the patient to raise his head up without support (lift head).
- Usually confirmed by palpating the defect between both recti.
- Causes: chronic increase of the intra abdominal pressure (HSM - ascites) + hypoproteinemia.

5. Umbilicus (4 Ss + CD)

Site	➤ Normal: midway between symphysis pubis and xiphisternum. ➤ Upper abdominal swelling and ascites slightly shift it downward, vice versa.
Shape	➤ Normal: Inverted. ➤ Everted umbilicus means chronic increase of the intra-abdominal pressure (HSM – ascities).
Swelling	➤ Hernia: expansile impulses with cough. ➤ Nodules: GIT of breast cancers (Sister Mary Joseph's nodule).
Skin lesions	➤ Scar. ➤ Wound.
Colours	➤ Cullen's sign: intra-peritoneal bleeding - acute pancreatitis " مهمة عملي " ➤ Causes of generalized pigmentation (see general ex VI).
Discharge	➤ Urine: patent urachus. ➤ Faces: colonic fistula. ➤ Pus: Suppurative inflammation. ➤ Bile: biliary fistula. ➤ Fluid: ascities (bursting abdomen).

6. Supra pubic hair distribution

- **Normal:** triangular in male with apex directed upward (umbilicus) & horizontal in females.
- **Abnormal:** as in
 - L.C.F.: feminine distribution.
 - Hypogonadism: loss of hair.

7. External genitalia

If the patient is circumcised - phemosis - para phemosis etc....

Inspection of the front "sides" (8)

1. Respiratory movement (see lung disease)

- Patient of tense ascites usually orthopneic and breath thoraco-abdominal.
- Patient of peritonitis usually has rigid dorsal decubitus and has absolutely thoracic breathing.

2. Breast: (one of the general examination items and must be examined in local abdominal examination).

- Gynaecomastia:** hyperplasia of the glandular component of male breast. Confirmed by pinching test as a disc (button like). Usually presented as a one sign of L.C.F. or caused by spironolactone. Comment on: laterality - tender or not.
- Breast atrophy** in females noticed in L.C.F.

3. Countour: (must be the first step) can be best appreciated by standing at the foot of the table and looking up towards the patient's head.

- **Normal:** slightly scaphoid with preserved waist.
- **Abnormal:**
 - Scaphoid:** the anterior abdominal wall is sunken and presents a concave rather than convex in Cachectic patient.

b. **Bulging.**

Symmetrical (5 Fs)	Asymmetrical
➤ Fat = obesity (sunken umbilicus) ➤ Flatus = (gases) ➤ Foetus ➤ Fluid = ascites ➤ Full urinary bladder	➤ Organ swelling (HSM) ➤ Ovarian or uterine tumor (comment on mass)

Comment on swelling

ملحوظة الكلام ده **advanced** شوية ،، صعب يتكلم معاك في حالة زي دي في الباطنة

ملما نشوف أي swelling نعلق عليها في ال history على الأتي

- Site مكان الكلكوعة دي فين ؟؟؟
- Size حجمها أد إيه ؟؟؟ يعني مثلاً في حجم ال lemon size ؟؟ في حجم ال orange size ؟؟
- Onset اكتشفت الكلكوعة دي إزاي ؟؟؟
- يعني accidental زي ال breast swelling

هل اكتشفت الكلكوعة دي acute onset ودي فيها sudden يعني خلال دقائق زي ال perforation أو rapid يعني خلال ساعات أو أيام زي ال acute inflammation

هل اكتشفت الكلكوعة gradual onset يعني خلال أسابيع أو شهور زي حالات ال chronic inflammation or neoplastic

- Course بتزيد ولا زي ما هي ؟؟؟
 - ✓ Progressive زي ال neoplastic swelling
 - ✓ Stationary زي ال chronic inflammation
 - ✓ Regressive زي ال inflammatory condition
 - ✓ Fluctuating زي ال chronic inflammation with acute exacerbation
- Duration بقالها أد إيه ؟؟؟
 - ✓ Short يعني بقالها أيام أو أسابيع زي ال inflammatory
 - ✓ Long يعني بقالها شهور أو سنين زي ال neoplastic
 - ✓ Since birth يعني من ساعة الولادة زي ال congenital
- Other swelling تسأل العيان هل فيه كلايخ ثانية عندك ؟؟؟
 - ✓ Multiple limpoma وال neurofibroma
 - ✓ Lymph nodes في الحالات ال inflammatory
 - ✓ Metastasis في ال malignancy
- Effect on the general condition يعني تسأل العيان سخنت ؟؟ اترعشت ؟؟؟ نفسك اتسدت ؟؟ خسيت جامد ؟؟؟
 - ✓ Toxic symptoms الي هي FAHM
 - ✓ Malignant symptoms الي هي cachexia
 - ✓ ال T.B. خلي بالك من ال night sweating, night fever, anorexia, emotional stress
- Apparent cause تسأل العيان : تفتكر إيه سبب الكلكوعة ؟؟؟
 - يعني مثلاً انخطبت ؟؟ شلت حاجة ثقيلة ؟؟؟ فيه حاجة نفسية تاغاك ؟؟؟
- What increase and what decrease ؟؟ في حاجة بتزودها أو بتنقصها ؟؟؟

ومتشاش تسأل عنها بردو تسأل كا past history و family history أكنه symptom عادي و complaint بتحله نفس الإستمبة المعتودين عليها يعني

لو جيت في ال examination نفس الإسكيم فيه عندنا في ال local examination بتاع ال swelling الأربعة الأساسيين : Inspection و palpation و Percussion و auscultation

لو جينا على ال inspection هنقول :

- Number ،، ال swelling ده يا ترى هل هو single ولا multiple
- Site ،، تقول ال anatomical site بتاع ال swelling ده
- Size ،، تقول هي كام في كام سم ،، وبتأخذ أكبر مقاسين يعني
- Shape ،، تقول يا ترى هي شكلها irregular ولا oval ولا rounded ولا butterfly زي الفراشة ؟؟؟
- Overling على ال skin الي بيكون swelling هنشوف
 - يا ترى هو الجلد ده طبيعي ولا مشدود stretched ؟؟ ولا واخد لون pigmented ؟؟؟
 - يا ترى عليه علامات ال Inflammation يعني لونه أحمر و مورم edematous ؟؟
 - يا ترى فيه dilated veins ؟؟؟ هل فيه ulcer ؟؟ هل فيه scar ؟؟؟
- وخلي بالك ،، في ال special signs ممكن نلاقيها مع ال swelling
 - hernia زي حالات ال Expansile impluse on cough
 - Pulsation زي ال aneurysm و ال vascular swelling
 - ببتحرك مع البلع زي ال thyroid
 - ببتحرك مع خروج اللسان زي ال thyroglossal cyst

لو جينا على ال palpation نخلي بالنا من كلمة تمسك دي TEMSC D

نشوف ال Temperature بتاعت العيان ،، ودي بنشوفها بظهر الإيد ،، وبنشوفها بالمقارنة عن طريق إننا نشوف جزء مفيهوش حاجة من الجسم ونقارنه بالجزء الي فيه ال swelling

نشوف ال **Tenderness** ، ودي بنشوها ببطن الإيد وعيني على وش العيان ، ممكن نلاقي ال swelling tender ال حالات ال inflammation غالباً بتكون tender وال neoplastic غالباً مبتكونش tender

نشوف ال **Edge** ، ودي بنشوها بجانب الإيد ونشوف هل ال well defined تعرف تطلع ليها edge و كده ولا الي هي ill defined

نشوف ال **Mobility** عن طريق تمسك ال swelling بإيدك وتحركها side to side و up and down

- وتشوفها هل هي skin attached ال skin ولا لا؟؟ لو مكنتش related ال overlying skin تقدر تعمل pinch ال skin لو كانت related ال skin متقدرش تعمل pinch ال skin
- تشوف إيه الي بيحصلها لما بيحصل muscle contraction
- هل بتبقا More prominent؟؟ يبقا دي superficial ال muscles
- هل بتبقا less prominent؟؟ يبقا دي deep ال muscles

طبعاً فيه تفاصيل أكثر يقولك ال nerves وال vessels وال tendons وال bones والكلام ده جراحة أكثر منه باطنة يعني ☺

نشوف ال **Surface** ، ودي حركة بنعملها براحة الإيد ، وبتمشي إيدك كده على ال swelling وتشوف يا ترى ال swelling ده smooth ولا granular ولا nodular ولا lobulated وهكذا

نشوف ال **Consistency** ، ودي حركة بكل الأيد ☺ ونشوف ال swelling دي يا ترى هي جامدة ولا ناشفة ولا حاجة مرنة كده وعادةً أي swelling عندنا حاجة من اتنين

- Cyst
- Solid ، وال solid ده بيتقسم إلى
 - ✓ Soft زي ال lobule of the ear
 - ✓ Firm زي ال tip of the nose
 - ✓ Hard زي ال forehead
 - ✓ Fleshy زي ال relaxed muscle

وفيه اختبارات بنعملها في ال cystic swelling ، زي ال fluctuation test وال paget test و ال bipolar test وفيه Modified fluctuation test وفيه ال crossed fluctuation test

بس أهم test هو ال fluctuation عشان كده هنجاول مع بعض نشرحه إن شاء الله

ال fluctuation test ده يا سيدي ، بداية تعرف إنه بيتعمل لو ال swelling الي ادامك دي cystic لو ال cystic swelling دي تحتوى على fluid هيظهرلك ال fluctuation وتحسه

المفروض إنك بتعمل ال test ده في مستويين متعامدين in 2 perpendicular planes

سيب صباعين عشان في ناحية يستقبلوا الموجة الي هتيجي ، وحرك بصاعين من الايد من الناحية المقابلة

نشوف ال **Draining lymph nodes**

وخلي بالك من الإن بي الي بتقولك ☺

No examination of a swelling is complete without the examination of the draining lymph nodes

وأخر شيء نشوف لو فيه special signs

زي ال pulsaation ، زي ال thrill ، هل فيه impulse on cough ؟؟ هل فيه reducibility زي ال hernia ، هل فيه compressibility زي ال saphena varix

percussion لو جينا على ال

لو عملنا percussion فوق ال swelling لو لقينا :

- Resonant زي الحالات اللي بتكون gaseous swelling زي ال herina لو كانت ال content بتاعتها Intestine
- Dull فوق ال cystic و solid swelling

نعمل percussion حوالين ال swelling عشان نحدد ال relation مع ال Organs اللي حوالها

auscultation لو جينا على ال

ممکن نلاقي الآتي :

- Systolic murmur زي aneurysm
- Machinery murmur زي A-V fistula
- Venous hum زي ال portal hypertension
- Intestinal sound زي ال hernia

الكلام ده ممكن يكون زيادات في الباطنة يعني ،، بس ممكن حد يبقا يكون Interested إنه يعرفه ،، وإن شاء الله يكون مفيد يعني لكل اللي يقرأه ونرجع ثاني لموضوعنا والنقطة الرابعة اللي هي ال Hernia

4. Hernial orifices

- Expansile impulses with cough.
- Preferred in standing patient.

متنصاش النقطة دي ،،

- لو دخلت على عيان عنده هرنيا " بس ده غالباً في الجراحة" لازم تقوله يقوم يقف عشان لو سبته نايم على السرير ممكن الممتحن يسقطك ☹
- Palpate the orifices defect.

بتشوف بإيدك الأماكن اللي بتخرج منها ال hernia ودي التقسيمات ال anatomical بتاعت ال hernia

1. **Epigastric** → small, midline through a defect in the linea alba located between the xiphoid process and umbilicus.
2. **Umbilical** → bulging defect at umbilicus.
3. **Incisional** → defect in abdomen muscles after surgical incision. Must palpate the size of the defect.
4. **Abdominal** → hernia through the abdominal wall.
5. **Inguinal** → Direct or Indirect.

5. Veins

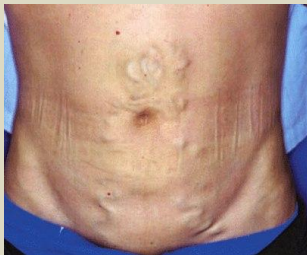
- *Is it visible or dilated ?*
- *If dilated. Is it systemic or portal ?*

a. Visible or dilated ?

- **Visible:** straight, narrow and not raised.
- **Dilated:** Tortuous, wide and raised above the level of the skin.

b. If dilated

Prominent, dilated veins may represent collateral circulation through the abdominal wall that has developed to compensate for obstruction of either the inferior vena cava or portal vein.

	Portal (caput medusa)	Systemic (IVC obstruction)
Site	Central	Peripheral
Milking test	Filling away from the umbilicus	From down to up
		

Milking test

- Place both index fingers closely, then sweep to the periphery.
- Allow for peripheral filling by releasing each finger.

Successively, direction of rapid filling is the direction of blood flow.

c. If systemic venous obstruction

S.V.C.	I.V.C.
Upper part	Lower part

6. Pigmentation

- Bluish at umbilicus: **Cullen's sign** (bleeding in peritoneum).
- Bruises on flanks: **Grey Turner's sign** (retroperitoneal bleeding as pancreatitis).
- Jaundice: yellow skin – usually due to liver disease or biliary obstruction.

7. Scratch marking

Denotes pruritis usually with obstructive jaundice

- Multiple, parallel and superficial.
- In accessible area.

ومناسبة ال obstructive jaundice « عشان تفتكر إيه اللي بيكون موجود فيها »

بنقول أربع كلمات : زيتونة بتهرش غسل وطحينة ☺

العيان بيكون لونه أخضر زيتوني « وبهersh « ولما يعمل حمام بيبقا ال urine غسل يعني dark
وال stool بيكون clay طحينة

ولما أفحصه general « ممكن ألاقى scratch marks ???

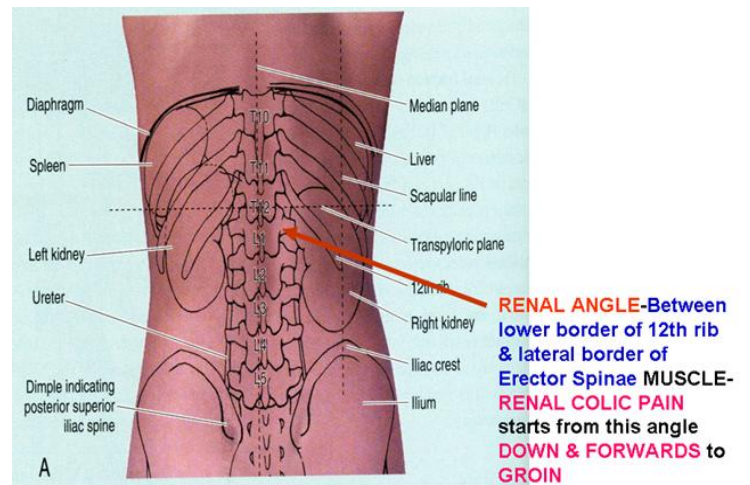
أه « بيهersh أكيد

8. Scars

- Surgical (name - healing).
- Traumatic.
- Cautary.

inspection of the back ولا تنسى أبداً

1. Scar.
2. Deformity.
3. Swelling of renal angle.
4. Cafe' au lait patches (neurofibroma).
5. Tuft of hair "spina bifida".
6. Renal angle "see kidney examination".



add for inspection in all views

- Scar:
 - Surgical (name - healing).
 - Traumatic.
 - Cautary.
- Stria.
- Scratch marking.
- S.C. bleeding.
- Pigmentation.
- Elasticity (dehydration).
- Oedema.

Palpation

General rules:

- Warm your hands.
- Ask patient if any part is tender: examine that last.
- Abdominal muscles must be relaxed by patient knee flexion.
- Superficial palpation then deep palpation.

Intra abdominal mass vs. abdominal wall mass:

- Ask the patient to raise his head.
- Mass of the abdominal wall remains palpable, where the intra abdominal mass will be obscured.

Types

a. Superficial palpation	b. Deep palpation
➤ To gain patient confidence. ➤ For tenderness or rigidity. ➤ Temperature. ➤ Superficial mass.	➤ For organomegaly. Or, ➤ Deep masses.

a. Superficial palpation

- Place your extended hand - flat on the patient's abdomen.
- Gently press the patient's abdomen by flexing the metacarpal joints (MCPs).
- Slowly progress around the 9 regions (either in S or G shape).



Assessment of muscle tone:

There are 3 reactions that indicate pathology:

- *Guarding: muscles contract as pressure is applied.*
- *Rigidity: rigid abdominal wall indicates peritoneal inflammation.*
- *Rebound: release of pressure causes pain.*

N.B.

- *If the patient is frightened, initially use the patient's hand under yours as you palpate. When patient calms then use your hands to palpate.*
- *The most sensitive indicator of tenderness is the patient's facial expression.*

Normal palpable structures:

- Sigmoid colon: left lower quadrant - firm, narrow tube.
- Cecum and ascending colon: right lower quadrant - a softer, wider tube.
- Pulsation's of ascending aorta: midline in upper abdomen.

Less commonly palpable, but normal:

- Liver: just below right costal margin (soft).
- Transverse and descending colon.
- Lower pole of the right kidney: (very deep, mostly in thin women).
- Iliac artery: pulsation's - left lower quadrant and right lower quadrant.

b. Deep palpation for

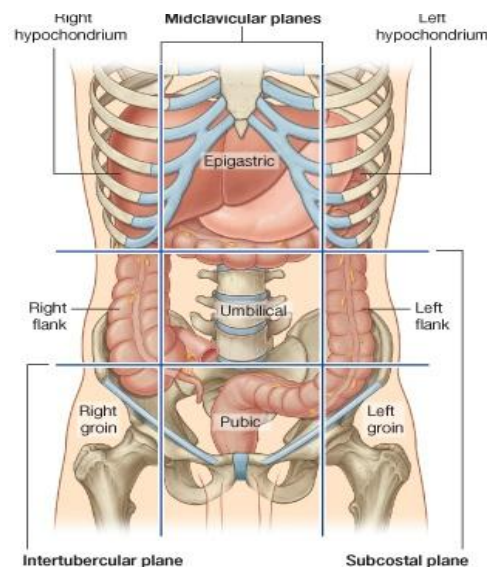
- Liver.
- Spleen.
- Kidney.
- Gall bladder.
- Urinary bladder.
- Colon and precolic mass.
- Lymph nodes and vessels.
- Inguinal and femoral areas.

Liver

Site: right hypochondrium and epigastrium.

Borders

Upper border	Lower border
➤ Lt - MCL 5th intercostal space. ➤ Rt - MCL 5th rib. ➤ Rt - MAL 7th rib. ➤ Rt - scapular 9th rib.	➤ Lt - MCL 5th intercostal space. ➤ Lt 8th costal cartilage. ➤ Midway between the xiphisternum & umbilicus. ➤ Rh 9th costal cartilage. ➤ Rt - MCL 1 inch below the costal margin. ➤ Rt - MAL 11th rib.



Liver palpation

Ordinary		Bimanual	Hooking	Dipping
Rt. Lobe at MCL from Rt. Iliac region	Lt. lobe at midline from umbilical region	As ordinary with Lt. hand support to back	If impalpable liver	In tense ascites

Liver palpation:

1. The upper border by percussion.
2. The lower border by:
 - Ordinary technique.
 - Tip of hands (Hutchinson's method).
 - Bimanual.
 - Hooking.
 - Dipping.
 - Ausultatory method (Macleod).

A normal liver is soft and not tender

➤ **Ordinary technique:**

- Right lobe at MCL beginning from right iliac region.
- Left lobe at midline from umbilical region.
 - ✓ Ask the patient to take a deep breath.
 - 1- By radial aspect of the index or tip of fingers.
 - 2- Feel the edge of the liver press against your fingers.
 - 3- Or, it may slide under your hand as the patient exhales.

➤ **Bimanual:**

- As ordinary with the left hand placed just below the right costal margin.

If impalpable liver

➤ **Hooking method:**

- Stand by the patient's chest.
- "Hook" your fingers just below the costal margin and press firmly.
- Ask the patient to take a deep breath.
- You may feel the edge of the liver press against your fingers.

➤ **Dipping:**

(for palpation of organs in tense ascites)

- Flex MCP joint fast to displace fluid and palpate a mass.

Disadvantage

- The organ descriptive details are impossible.

N.B.

- Lower border can be detected by
 - 1- Light percussion.
 - 2- Stethoscope. (scratch).

Characters of hepatic swellings:

- Intra- abdominal swelling i.e. moves up & down with respiration.
- At Rt. Hypochondrial region.
- Sharp borders, firm consistency & smooth surface.
- Not tender & not pulsating.

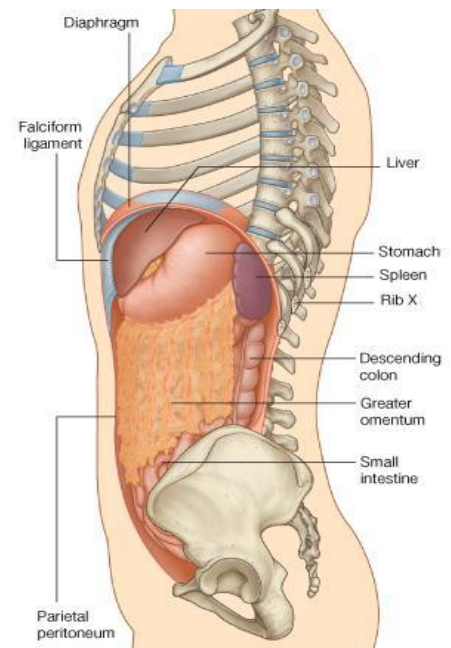
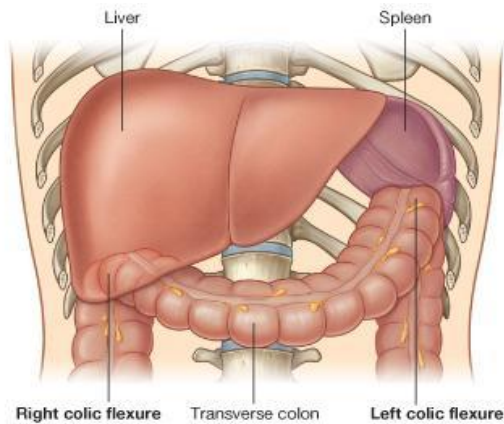
Causes of hepatomegaly ???

Spleen

Surface anatomy of the spleen

Site: left hypochondrium

- Under 9th, 10th & 11th ribs (long axis on 10th rib).
- Medially: scapular line.
- Laterally: MAL.



Spleen palpation

Ordinary	Bimanual	Hooking	Dipping
From Rt. Iliac fossa toward Lt. costal margin	With left hand support + Rt. Lateral position if impalpable	If impalpable spleen	In tense ascites

Spleen palpation:

- Ordinary technique.
- Tip of hands.
- Bimanual.
- Right lateral decubitus.
- Hooking.
- Dipping.
- **Bimanual spleen palpation:**
 - Support lower left rib cage with left hand, while patient is supine and lift anteriorly on the rib cage.
 - Palpate upwards from the right iliac fossa toward the spleen with finger tip of right hand.
 - Ask the patient to take a deep breath.
 - Palpate for spleen as it descends.
 - A palpate spleen is almost always abnormal.



Spleen enlarged toward the patient right iliac fossa due to attachment of phrenico-colic ligament.

عشان كده مهم جداً لما تيجي تفحص ال spleen تبدأ من ال right iliac fossa عشان دا ال direction بتاع ال spleen لما يكبر ومتنشاش بسبب ال phrenico-colic ligament

But, spleen may be directed to left iliac fossa if:

- Huge spleen.
- Absent ligament congenitally or surgically removed.
- Infiltration of the ligament by malignancy.

Comment on

	Liver	Spleen
Consistency	Firm	Firm
Border	Sharp	Sharp
Size	Patient fingers	Patient fingers or huge
Surface	Smooth	Smooth
Tenderness	Not	Not
Pulsation	Not	Not
Notch	---	Notched
Pitting	---	not

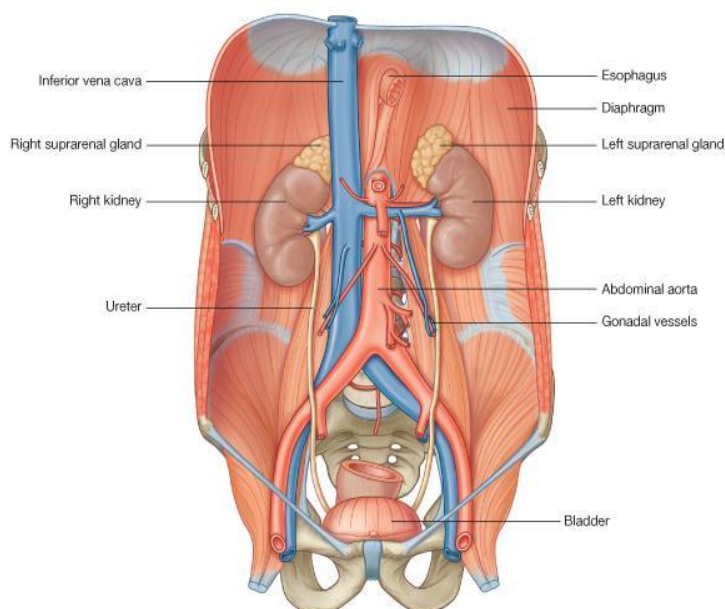
Splenomegaly

Just palpable	Moderate	Huge
➤ S.B.E. ➤ Salmonella ➤ Sarcoidosis ➤ Septicemia ➤ T.B. ➤ I.M.N.	➤ Lymphoma ➤ Leukemia ➤ Portal H⁺⁺	➤ Bilharziasis ➤ Thalassemia ➤ Myeloid Leukemia ➤ Kala Azar (not in Egypt) ➤ Polycythemia V. ➤ Sarcoma

Kidney

Anatomy

- **Size:** 12 X 6 X 3 cm.
- **Position:**
 - The kidney is retroperitoneal occupying the loins.
 - They are in the para-vertebral gutter.
 - The right kidney is lower than the left by 0.5 inch because of the pressure of the liver on the right side.



	Rt. Kidney	Lt. kidney
Upper pole	Lower border of T ₁₂	Upper border of T ₁₂
Lower pole	Lower border of L ₃	Upper border of L ₃

Surface anatomy of the kidney

- a. **Posterior surface marking of the kidney:** bounded by 4 lines (**Morris' parallelogram**):
 - 2 vertical lines: 3 & 9 cm from median plane.
 - 2 horizontal lines: at level of T₁₁ and L₃.
- b. **Anterior surface marking of the kidney:**

	Rt. Kidney	Lt. kidney
Upper end	11 th space	11 th rib
Lower end	5 cm above iliac crest	6.5 cm above iliac crest

Kidney examination

a. Inspection

- As usual + sinus.
- Swelling:
 - ✓ Fullness → renal mass (inside capsule).
 - ✓ Bulge → perinephric or tumor.

b. Palpation

- **Bimanual:** lower pole of right kidney.
- **Ballotement.**
- **Capture.**

c. **Percussion:** "Dullness - tenderness"

d. **Auscultation:** renal artery stenosis.

Character of kidney swelling:

- Intra abdominal.
- Moves up & down with inspiration (limit).
- At lumbar region.
- Reno-from.
- Fills renal angle.
- Can insinuate our hand above it.
- Band of resonant.
- Posterior balloattment.

➤ **Bimanual technique:**

On supine position:

1. **Right kidney** (stand on the right side of the patient).
 - Put your finger tips of the left hand below 12th rib (costo-vertebral angle), then lift up.
 - Press the right hand below costal margin and try to capture the kidney, then ask patient to breath.
2. **Left kidney** (do the reverse) stand on left, left hand up and right below.

وخلي بالك عشان متنساش وتفصل واقف على شمال العيان لما تيجي تعمل bimanual technique ،، سيب الورق بتاعك وشنطتك على يمين العيان عشان تفكر إنك ترجع لمكانك الطبيعي في الإمتحان ،، اللي هو على يمين العيان

➤ **Ballottement method:**

- Keep your anterior hand steady in the deep palpation position in the right upper quadrant lateral and parallel to rectus muscle.
- Attempt to ballot the kidney with the other hand in renal angle.
- An enlarged kidney should be palpated by the anterior hand.
- Repeat the same maneuver for the other kidney.

Normal: the kidney are not usually palpable, except the inferior pole of the right kidney.

Abnormal finding:

If mass is identifiable, note:

- **Size:**
 - Unilateral enlargement: (hydronephrosis, tumor, cyst).
 - Bilateral enlargement: (polycystic kidney, lower urinary obstruction).
- **tenderness**
 - acute pyelonephritis.
 - Acute glomerulonephritis.

Technique of renal angle examination

- Patient sitting position.
- Gently press the angle.
- Then percuss it.
- Feel the paravertebral muscles.
- Assess tenderness of ribs, paraspinal muscle and spine.
- Auscultate the costovertebral angle for bruit.

Caution:

- Forceful blow will elicit tenderness even in normal patients. Be gentle.
- Tenderness of the ribs, vertebra or paraspinal muscles is usually not of renal origin.

Abnormalities of costovertebral (renal angle)

- Renal angle tenderness:
 - ✓ Acute pyelonephritis.
 - ✓ Acute glomerulonephritis.
 - ✓ Renal or peri-renal abscess.
 - ✓ Acute hydronephrosis.
- Bruit:
 - ✓ A-V fistula.
 - ✓ Renal artery stenosis.
 - ✓ Vascular tumor.

D.D. between

Splenic swelling	Left renal swelling
I can't	I can insinuate my finger between costal margin & the swelling
Notch (pathognomonic)	No
Empty renal angle	Full
No posterior ballottement	Positive posterior balloattement
Surface: smooth	Rounded
Edge: sharp	Bossy
No resonant	Band of resonant

Spleen: continuous with splenic dullness (Traub's area).

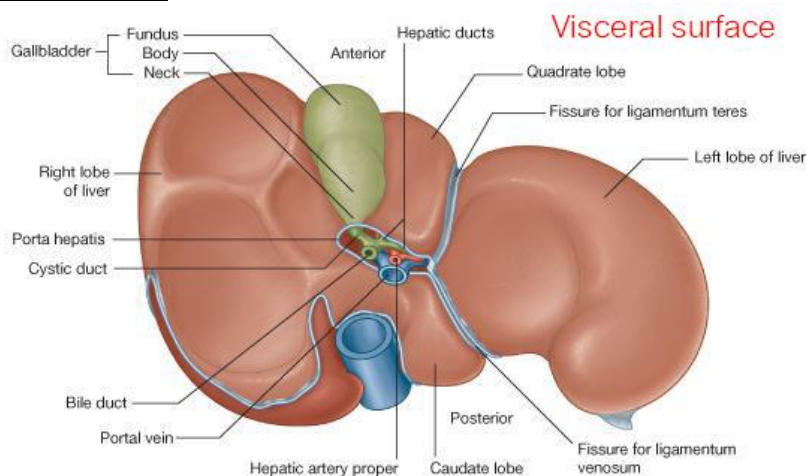
Gall bladder

Anatomy

- **Shape:** pyriform-shaped.
- **Size:** 8 - 12 cm length X 3 cm width.
- **Site:** fossa for gall bladder on the inferior surface of the right lobe of the liver.
- **Capacity:** 30 - 50 ml.
- **Power of concentration:** 10 times.
- **Parts:** funds, body and neck.
- **Nerve supply:**

- **Autonomic:**

- ✓ Parasympathetic: hepatic branch of anterior vagus.
- ✓ Sympathetic: from T₇₋₉ (pain is referred to inferior angle of right scapula).



حتى apply نستفيدها هنا ، في موضوع ال Peptic ulcer ، وأدليك Hint بسيط عن موضوع ال Peptic ulcer
 ال peptic ulcer ده أشهر حاجتين منه ، ال duodenal ulcer وال gastric ulcer
 ال Ulcer أصلاً في تعريفها ما هي إلا loss of continuity of mucosa
 والسبب عدم التوازن imbalance يعني بين ال Mucosa وال HCL

يعني في حالات ال gastric ulcer غالباً فيه ضعف في ال Mucosa ، فال HCL لو قليل بيكون نسبياً أعلى على ال gastric mucosa فيبعملنا gastric ulcer

وفي ال duodenal ulcer بيكون المشكلة إن فيه hyper acidity شويتين

ففي العلاج بتاع ال gastric ulcer بنلجأ إلى ال medical treatment وننتظر لل healing بتاع ال Ulcer
 بس المشكلة لو العملية طولت ، بنخاف إنها تقلب malignancy ، فبتدخل جراحياً جامد ، ونعمل gastrectomy ونشيل ال Ulcer بالمره وبنعمل biopsy

في ال duodenal ulcer بزوح على ال medical treatment ، ونستنى شويتين ثلاثة أطول من ال gastric ulcer
 المهم ، لو فشل العلاج ال medical ، بنعمل حاجة اسمها vagotomy
 إننا نقطع ال vagus ، عشان ال vagus ده اللي بيغذي ال parietal cells اللي بتطلع ال HCL ، وال HCL عندنا عالي
 فإحنا لما بنعمل vagotomy ده بيخلي نسبة ال HCL قليلة
 طبعاً عندنا في ال vagotomy أكثر من طريقة ليها ، فيه منها ال truncal vagotomy ونشيل ال anterior vagus وال Posterior vagus
 وفيه طبعاً طريقة أخرى زي ال selective vagotomy وفيه ال highly selective وفيه seromyotomy

الشاهد في الموضوع لما بنعمل truncal vagotomy ونشيل ال anterior vagus ، ده بيأثر على ال Innervation of the gall bladder ، فال gall bladder متعرفش تنقبض كويس وتعملي biliary dyskinesia
 و gall stones وحارات ☺

- **Sensory:** by the right phrenic C3,4,5 (the same segments as the supra clavicular nerves → so, pain in gall bladder is referred to right shoulder).

Character of the gall bladder:

- Pyriform - cystic.
- Mobile from side to side.
- Attached to the liver.
- Dull.

D.D.

Reidl's lobe (accessory lobe of the liver).

Causes of enlarged gall bladder

- Painless = neoplasm.
- Painful = mucocele - pyocele - torsion peri-cholecystic abscess.

Murphy's sign

(Benjamin Murphy 1857 - 1916)

Technique:

- Firstly ask the patient to breath out.
- Then gently place the left hand below the costal margin on the right MCL.
- Lastly, ask the patient inspire (breath in).
- Positive test: the patient suddenly hods breathing.

Courvoisier's law : Stones = stays small since scarred

Urinary bladder

- Ask the patient when last urinated, and whether was complete emptying.
- Usually palpable if full, usually not palpable if empty.
- Look for palpable, empty bladder (swelling).

Character of U.B.

- Pelvi-abdominal (has no lower end).
- Ovoid.
- Cystic.
- Immobile.
- Dull
- Positive fluid thrill.
- Pressure = desire of micturation.

Colon

- Palpable in I.B.S. by rolling.
- Ch.Ch.
 - Cord - like.
 - Move side to side.
 - Gargling sensation.



Pericolic masses

D.D. right iliac fossa mass

- Pericolic mass of Bilharziasis.
- Cancer colon.
- L.N.

Aorta - L.Ns.

Longitudinal line from xiphsternum by deep rolling.

Percussion

Simple roles

- The percussing finger: middle finger of the right hand.
- The movement: from the wrist joint.
- The percussed finger: middle finger of the left hand

Abdominal percussion:

1. Ascites.
2. Liver borders: liver span.
3. Spleen: for splenomegaly.
4. Kidneys.
5. Bladder: for enlarged bladder or pelvic mass.
6. Masses.

1. For ascites

Mild	Moderate	Severe or tense
Knee elbow position	Shifting dullness	Transmitted thrill
50 - 1500	1500 - 3000	> 3000

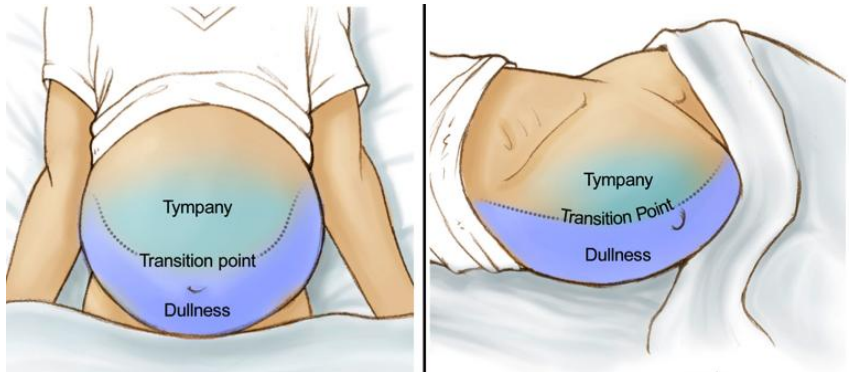
Technique**First step:**

Direct percussion is done over the abdomen, from the umbilicus to the flanks.

- The location of transition from tympany to dullness is noted.

Ascites: percussion note is tympanitic over the umbilicus and dull over the lateral abdomen and flank areas (U shape dullness).

Note: the tympany over the umbilicus occurs in mild or moderate ascites, because bowel floats to the top of the abdominal fluid.

**Shifting dullness**

Percuss from the midline to the flanks on both sides:

- Place the finger parallel to the expected edge.
- Percuss from resonance in the mid-abdomen to dullness in the flanks.
- The patient rolled to the opposite side.
- Waiting for 30 seconds.
- The previous area of dullness should now be resonant.

It does not matter which side one chooses to start with.

Transmitted thrill (fluid thrill)

- Place one hand on the patient flanks.
- With the other hand briskly tap the other flank.
- A third hand is placed in the mid-abdomen with sufficient pressure applied to dampen any wave that may pass through the anterior abdominal wall.

Positive test: a shock wave be felt with palpating hand.

**Knee elbow position**

- Place the patient in knee elbow position and percuss the umbilicus area.

2. Liver

For detection of the liver span

Liver span:

- The distance between both upper and lower borders of the liver in the right MCL.
- About 8 - 12 cm in a normal adult.

The upper border (heavy percussion)

Percuss downward from the chest in the right mid clavicular line until you detect the top edge of liver dullness.

The upper border of the liver is normally in the right 5th intercostal space.

The lower border:

By palpation (see above) or light percussion. Percuss upward from the abdomen in the same line until you detect the bottom edge of liver dullness.

N.B.

- *an alternate method of estimating liver size is the scratch test.*

3. Spleen

Percussion of the spleen is more difficult as this structure is smaller and lies quite laterally under completely under ribs.

Percussion of the spleen:

- The two most useful methods are percussion over Traub's area and Castell's sign.
- Percussion should be carried out at one or more levels of Traub's area from medial to lateral (see lung diseases Volume 2).

Castell's method

Technique

- Percuss the lowest intercostal space (8th or 9th) in the left anterior axillary line.
- With the patient in full inspiration and then full expiration.
- Positive sign: if the note changed from resonant on full expiration to dull on full expiration.

(when the patient inspires, the spleen moves inferiorly along the posterolateral abdominal wall. If the spleen is enlarged enough that the inferior pole reaches the 8th or 9th intercostal space).

4. Urinary bladder

Percuss the upper border of the urinary bladder

- In 3 longitudinal lines (the midline and 2 para-median lines).
- The upper border of the urinary bladder (or any pelvic swelling) noticed as convex (n shape) line.

Summary of general value of percussion:

- Gaseous distention of bowel (intestinal obstruction - IBS): diffusely tympanic.
- Solid mass (e.g. HSM): dullness over the most protuberant part and tympanic in the flanks.
- Ascites: tympanic in the central portion and dull in the flanks.

Auscultation

Auscultation of the abdomen has a relatively minor role

For,

1. Intestinal sounds.
2. Vascular sounds.
3. Scratch test.
4. Succession splash.
5. Buddle sign. مهمة سؤال شفوي على حالة
6. Friction rubs.

Pre - warm diaphragm of the stethoscope by rubbing it on the front of your shirt before beginning auscultation.

1. Intestinal sounds

The diaphragm of the stethoscope should be placed on the abdomen, as least initially in the right lower quadrant near the ileocecal valve.

Specifically listen for the pitch & frequency of the bowel sounds.

Normal: gurgling, 5 - 35 per minute.

Rushing (Borborygmi): loud and easily audible - normal in diarrhea.

على فكرة، ال Borborygmi ده الصوت اللي بيطلع، وليس اسم لعالم

Tinkling: high pitched, (raindrops in a barrel): intestinal obstruction.

Decrease sounds: (none for a minute) - decrease gut activity.

After abdominal surgery, abdominal infection (peritonitis) or injury.

Absent sounds: no sounds for 3 minutes. **Bad sign** ☹️ ➔ ileus.

2. Vascular sound

	Arterial bruits	Venous hum
Timing	Only during systole	Continuous
By	Diaphragm	Bell
Chr.	High - pitched	Low - pitched
Increased by	---	Inspiration

a. Venous hum

Best heard overlying the portal vein in an area approximated by an elliptical shape between the umbilicus and the mid-clavicular line where it crosses the right subcostal margin.

b. Arterial bruits

Aorta	Begins at or just to the left of the xiphoid process and bifurcates at the level of the umbilicus.
Renal a.	Midway between the xiphoid process and the umbilicus and within 2 cm of the midline.
Bifurcation of CIA	Mid-way between the umbilicus and the mid inguinal point (MIP).
Femoral	Just above the inguinal ligament (MIP)

N.B.

- Blood flow through the aorta itself usually does not generate any appreciable sound.

3. Scratch test**Technique:**

- Place the diaphragm over the area of the liver.
- Then scratch parallel to the costal margin until the sound intensity drops of marking the edge of the liver.

Different patterns of scratch may be used (e.g. spokes of a wheel).

4. Succession splash.

Called auscultation of the stomach

Detected in gastric outlet obstruction e.g. pyloric ulcer or neoplasm.

Technique:

- Place the stethoscope on the epigastrium.
- Then shake both iliac crests.
- While shaking, listen to splash from retained fluid. خبط حول السماعه

5. Buddle sign

- In knee elbow position.
- It is useful for detecting small amounts of ascites as small as 120 ml (shifting dullness and bulging flanks typically requires 500 ml).

6. Friction rubs (rare)

- Right and left upper quadrant.
- Grating sound with respiratory movement.
- Indicates inflammation of peritoneal surface of an organ.

Auscultation over the liver:

- Friction rub (grating during breathing) peritonitis.
- Bruit (cancer).
- Scratch.

Auscultation is sometimes done before percussion and palpation, unlike in other examination. 😊

- It may be performed first because vigorously touching the abdomen may disturb the intestines, perhaps artificially altering their activity and thus the bowel sounds.
- Additionally, it is least likely to be painful - invasive; if the person has peritonitis and you check for rebound tenderness and then want to auscultate you may no longer have a cooperative patient.

Value of P.R.:

- **Sphincter:** Loss of tone and patulous. (Cauda equina syndrome).
- **Contents:** Hard impacted stools, Foreign body.
- **Rectal wall:** Pelvic masses (Ovary, Uterus) in women.
- **Mucous membrane:** Irregular, Mass. (Cancer), piles (complicated).
- **Prostate** in men:
 - Smooth, large, firm and non-tender. (Benign enlargement)
 - Hard, irregular nodule or fixed hard mass. (Cancer, Stone, Chronic prostatitis).
 - Large, boggy and tender. (Acute Prostatitis).
- **Stools:**
 - Bloody: (Hemorrhoids, Bleeding rectal lesion).
 - Black: (Upper GI bleed, Iron, Some Antacids).

Diagnosis

Aetiological	Anatomical	Functional
1. Malignant 2. Cirrhosis 3. Mixed ↓ • History. • Investigations	1. Organo-megaly (liver - spleen) 2. Ascites ↓ by • Local examination • Ultra sound	• Compensated. Or, • Decompensated a. Vascular. b. Parenchymatous ↓ • History Haematemesis Encephalopathy • General examination

N.B. in Bilharziasis or mixed type

Staging

- i. Hepatomegaly.
- ii. Hepatomegaly + splenomegaly.
- iii. Shrunken liver + splenomegaly.
- iv. Ascites.

Premature ascites

Definition: ascites before stage IV.

Aetiology: local causes (e.g. T.B. peritonitis - malignancies - SBP).

Diagnosis

A case of HSM for D.D., most probably due to Bilharziasis compensated and not complicated.

Abdomen (long case)

A. History

Personal history

Female patient, xxx, 47 years old, from Al-Fayoum, house wife, married 29 years ago and has 3 sons, the youngest is 19 years old, she has no special habits of medical importance.

Complaint

She is complaining of abdominal swelling of 2 weeks.

History of present illness

The condition started 15 years ago by an active attack of haematemesis of sudden onset with 2 cups of dark brown blood preceded by nausea and followed by melena for 3 days, she admitted in Aldemerdash hospital, investigated by upper endoscopy & treated by endoscopic sclerotherapy without blood transfusion and this attack is not recurrent until now.

- No appetite changes, dysphagia, heart burn or halitosis.
- No changes in bowel habits or bleeding per rectum.
- 10 years ago, patient suffered from lower limbs oedema with gradual onset and progressive course, responds to diuretics.
- Followed after 2 years by ascites with gradual onset and progressive course with several paracentesis process of about 3 liters of clear fluid and not followed by any complications.
- Lower limb oedema & ascites are associated with jaundice and pruritis with dark urine and low grade fever & progressive mild loss of weight.
- No abdominal pain or urinary symptoms.
- No gynaecological symptoms.
- No symptoms of other system affection.
- Patient is investigated by CBC, liver function tests and abdominal ultrasonography then treated by vitamins & diuretics.

Past history

History of bilharziasis at age of 13, manifested by dysentery, investigated by urine & stool analysis and treated by praziquantil.

- No history of other chronic diseases, surgical operations or drug intake.

Family history

Irrelevant.

B. General examination (very important)

- Patient with an average general condition, average built.
- Patient is fully conscious, oriented for time, place and persons
- Patient lies in semi sitting position.

Head and face examination

- Orange yellow jaundice, wasted temporalis, with endemic parotitis.
- No pallor or cyanosis.
- No puffy eyelids with normal eye brows.
- Bad oral hygiene with normal tongue size.

Neck examination

- Congested pulsating neck veins, JVP = 4 cm above sternal angle with normal waves.
- Palpable carotid pulsation without thrill.
- Central trachea - no lymph node enlargement or thyroid swelling.
- Spider nevus in root of the neck.

Upper limb examination

- **Pulse:** 75 beats / minute, regular, big volume,
- **Blood pressure:** 130 / 50 mmHg.
- **Palmar erythema:** are present in both hands.

Lower limbs examination

- Shows bilateral, painless, pitting oedema.
- **Echymotic patches:** are present in lower limbs, upper limbs and trunk.
- **Systems examination:** no abnormalities were detected are regard C.V.S., chest & C.N.S. examination.

C. Local abdominal examination

Inspection

- Symmetrical enlargement of the abdomen with full flanks, wide subcostal angle, and abdomen moves freely with respiration.
- No gynecomastia, epigastric pulsation.
- There are divercation of recti, umbilicus is shifted downward, everted with umbilical hernia, no pigmentation, discharge or nodules.
- No stria, scan other hernia or dilated veins.
- No swelling in renal angle or back deformity.

Palpation

A. Superficial

- No superficial swelling or tenderness.

B. Deep palpation

- Right & lower lobes of liver are not palpable.
- Spleen: swelling in the left hypochondrium intra-abdominal, 5 fingers below the left costal margin with notch, sharp edge firm in consistency, smooth surface, not tender & not pulsating.
- Kidney are not palpable.
- No any palpable mass.

Percussion

- Moderate ascites is detected by shifting dullness.
- Dullness on Traub's area.

Auscultation

- Normal intestinal sounds.
- No vascular sounds, rubs or splachs.

Diagnosis

A case of shrunked liver with splenomegaly for D.D. most probably due to mixed bilharzial & hepatitis cirrhosis, with vascular and cellular decompensation.

Rheumatological Sheet & Examination

Introduction :

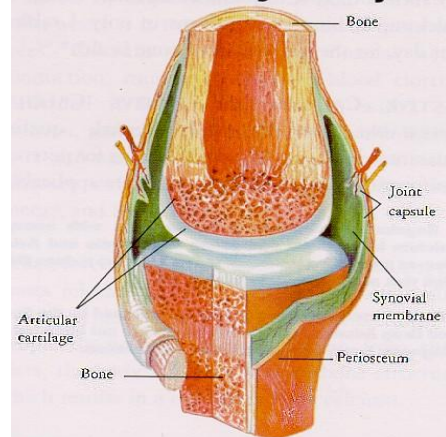
- **Arthralgia:** Pain arising in joints without signs of inflammation (ie. No limitation of movement).
- **Arthritis :** Joint with redness, hottness, swelling & limiting of movement
- **Mono arthritis:** One joint inflammation.
- **Oligo (pauci)arthritis :**2- 4 joints inflammation
- **Poly arthritis:** affection of more than 4 joints.
- **Bursitis:** inflammation of bursa (with Rhd. A.).
- **Enthesopathy:** inflammation at points of muscle & tendon insertion (in sero – ve Arthritis).
- **Myositis:** inflammatory disease of the muscle.
- **Synovitis:** Synovial joint inflammation.
- **Teno Synovitis:** Tendon sheath inflammation.
- **Centripetal arthritis:** polyarthritis starts at peripheral small joints (inrpharyngeal, metacarpopharyngeal or wrist) then spreads proximally to involve a larger joint (knee, hip or axial) **e.g. Rh. A.**
- **Centrefugal arthritis:-** polyarthritis starts at a large joint (knee, hip or axial) then spreads proximally to involve peripheral small joints (inrpharyngeal, metacarpopharyngeal or wrist) **e.g. Ank. spon**

Joints

1. *synovial joints:*

- *freely movable*
- *Separated by articular cartilage & synovial cavity.*
- *Lubricated by synovial fluid.*
- *Surrounded by a joint capsule e.g.: knee & shoulder.*

Structure of a synovial joint

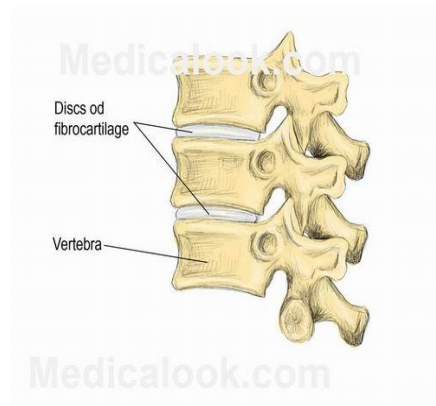


2. *cartilaginous joints:*

- *Slightly movable.*
- *Contain fibro cartilaginous discs that separate the bony surface.*
- *Have central nucleus pulposus & annulus fibrosus e.g.: vertebral discs.*

3. *fibrous joints:*

- *No appreciable movement.*
- *Consists of fibrous tissue or cartilage.*
- *No joint cavity e.g.: skull sutures.*



History

- **Personal history.**
- **Complaint.**
- **H.P.I.**
 - A. General.
 - B. Articular.
 - C. Extra articular.

A. General

(Prodromal manifestation)

(Fever- Headache - Hmalaise - anorexia - weight loss)

B. Articular

1. **Pain.**
2. **Swelling.**
3. **Stiffness.**
4. **Deformities.**
5. **Disability.**

1. pain

- هل مفاصلك فيها ألم؟
- يبيزيد بالراحه ولا بالمجهود؟

You have to diffrentiate between:

1. Articular & non articular pain.
2. If articular (inflammatory or mechanical).

Articular	Non articular
➤ Tender joint. ➤ Disturbe active &passive movement. ➤ Deformity.	➤ Tenderness outside joint lines. ➤ Passive movement is better than active. ➤ No deformity. (As bursitis & tendonitis).

Inflammatory	Mechanicalcal
➤ Increased by rest. ➤ Decreased by activity. ➤ More in morning.	➤ Increased by activity. ➤ Decreased by rest. ➤ More at night.

N.B.

- **Poorly localized hand & wrist pain .**
Numbness + night pain = carpal tunnel syndrome.
- **Vertebral pain:**
 - Upper: rheumatoid arthritis.
 - Lower: ankylosing spondylitis.

2. swelling

- هل مفاصلك ورمت؟
- أي مفصل؟
- هل الورم مستمر ولا يروح ويبجي؟

Causes:

- Joints (signs of inflammation).
- S.C. nodules.

3. Stiffness

(morning stiffness) M.S.

- مفاصلك مخشبه عليك الصبح؟
- لمدة أد إيه؟

Significant M.S. if >30 min.

But may improve by treatment so history of drugs intake is important.

4. Deformities

- هل حصل تشوهات في اي مفصل؟

5. Disability

(functional diagnosis)

- هل أثرت علي شغلك ولا لأ؟

C. Extraarticular history

- Rheumatoid arthritis.(mild)
- S.L.E.: marked(sever).
- Osteoarthritis:(No).

a) C.V.S.:

- Palpitation (A.R).
- Pain (pericarditis).
- Thromboembolic manifestation.

b) Respiratory system:

- pain (pleurisy - effusion).
- Cough.
- Dyspnea (I.P.F.).

c) C.V.S.:

- Convulsion.
- P.N. - pain.
- Weakness.
- Muscle pain.
- Stroke.

d) Abdomen:

- Dysphagia.
- Abdominal pain.
- Kidney (L.L. oedema - urine changes).

e) Skin:

- Alopecia.
- Rashes.
- Orogenital ulcers.
- Urticaria.
- Pigmentation.
- Cold peripheries. (Raynaud's).

f) L.N. :

- Swelling.

g) Eyes:

- Dry, red eyes.
- Pain.
- Photophobia.

Examination

A. General

B. Extra-articular

C. Articular

A. General

- Temperature, pulse {priphral pulsation for vasculitis }.
- Blood pressure for vasculitis.
- Face for cushinoid face (steroid therapy).

B. Extra-articular

a. **C.V.S.:** Effusion - H.F. - A.R.

b. **Respiratory:** Effusion - Crepitation (I.P.F.).

N.B.:

- Caplan's syndrome (R.A. - lung nodules - pneumoconiosis)

c. **C.N.S.:**

- P.N.
- Myopathy (from steroid therapy).
- Pyramidal tract lesion (compression).

d. **Abdomen:**

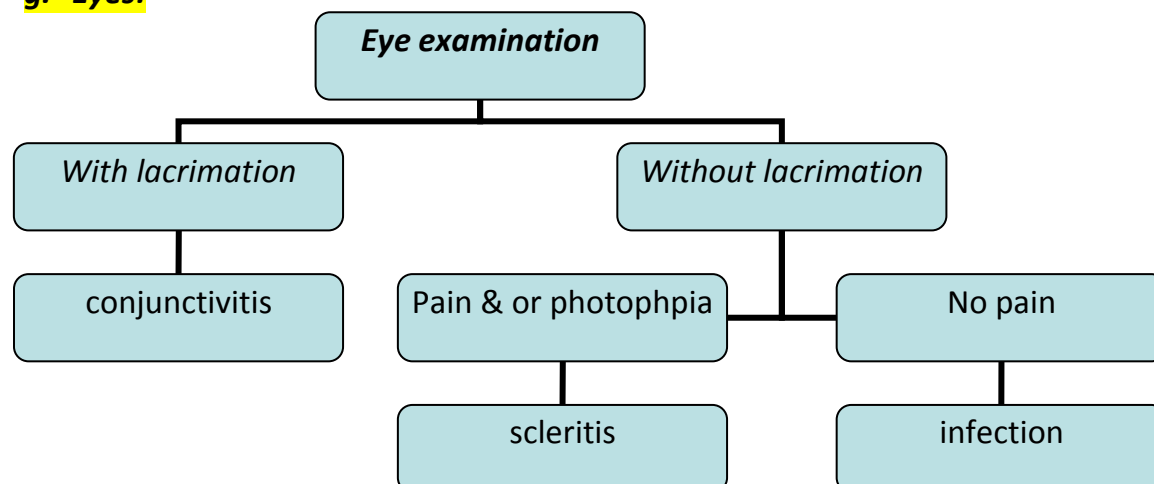
- H.S.M. (felty's syndrome & still's disease).

e. **Skin:**

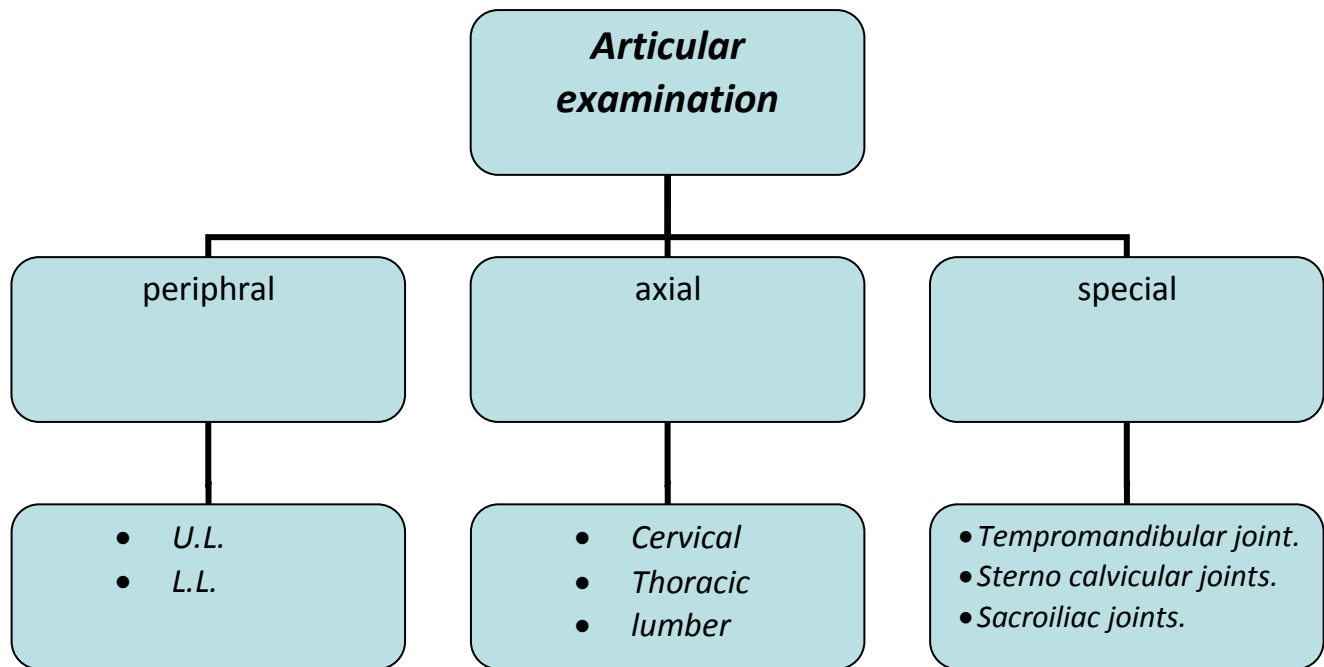
- Alopecia.
- Raynaudes.
- Nail fold infarction (psoriasis).

f. **L.N.:** +++++

g. **Eyes:**



C. Articular



Peripheral joints

Compined inspection & palpation:



8 points: (SM SM CD TT)

1. **Skin**: look for erythema, warmth, ulcers, sinuses or scar.
2. **Muscle**: look for wasting (thenar, hypothenar,)
3. **Swelling**: effusion, synovium, bony, SC nodules.
4. **Movement**:
 - Range of movement.(active - passive).
5. **Crepitus**.
6. **Deformities**:
 - Describe it :
 - Ulnar deviation (MCP)
 - Swan neck.
 - Button hole.
 - Z shaped thumb.

N.B.

- Deformity may be correctable if in soft tissue or in correctable in bone & cartilage.

7. **Tenderness**: joints or surrounding structures.**Grades**

1. Patient says. (يقول)
2. Patient winces. (يصرخ)
3. Patient withdraws. (يسحب)
4. Patient refuse joint touch. (يرفض)

8. **Special Test**: as Carpal Tunnel syndrome**Hands & Wrist**

1. **Skin**: palmer erythema & nails.
2. **Muscles**: wasting
3. **Swelling**:
 - Effusion:
 - IPJ : sausage.
 - MCPJ: knuckles.
 - Synovium: gritty sensation around joint.
 - Bony :
 - Heberden (distal).
 - Boucher (proximal)
 - S.C. nodules.
4. **Movement**:
 - Hand grip.
 - Finger :flexion - extention - Abduction - Adduction.
 - Wrist : Extention - flexion.
5. **Deformation**:
 - ulner deviation.(MCP)
 - radial deviation.(wrist)
6. **Tenderness : Grade & site .**
 - I.P.J.: side to side.
 - M.C.P.J.: squeezing method or by 2 thumbs.
 - Wrist: squeezing method or by 2 thumbs.

Tests in carpal tunnel \$

- ✚ **Tinnel's sign**: percussion over the carpal tunnel elicits tingling in the affected finger.
- ✚ **Phalen's sign**: forced flexion of the wrist over 30 seconds elicits tingling.
- ✚ **Examine the thumb abduction.**

Elbows & R.U.J

1. **Skin:** erythema
2. **Muscles:** wasting.
3. **Swelling:**
 - Detected in posterior triangle between 2 epicondyles & olecranon.
 - S.C. nodules.
4. **Movement:**
 - Elbow: full flexion & extension .
 - R.U.J.: with shoulder adduction & elbow flexion do supination & pronation.
5. **Crepitus:**
 - Elbow: *palpation with flexion & extension.*
 - Radius head: *palpation with pronation & supination.*
6. **Deformity:**
 - Flexion deformity.
 - Integrity of collateral ligaments with fully extended elbow.
7. **Tenderness:**
 - Grade.
 - Site :
 - Over grooves overlying the joints (arthritis).
 - Over lateral epicondyle (Tennis elbow).

Shoulder (acromio-clavicular joint)

1. **Skin:** erythema.
2. **Muscles:** wasting.
3. **Swelling:** No.
4. **Movement:** (R.O.M)
 - Abduction to 90° with palms to downward.
 - Raise vertically with palms facing others.
 - Place behind neck (abduction + external rotation).
 - Place behind back (adduction + internal rotation).
5. **Tenderness :**
 - Site .
 - Grade.

N.B.: Rotator cuff \$

-  Supraspinatus tendonitis.
-  Pain on start of abduction & when passively abduct till 45°.
-  Patient can continue abduction without pain by deltoid.

Ankle & feet

1. **Skin:** calluses.
2. **Muscles:** wasting (as C.N.S)
3. **Swelling:** S.C. nodules at tendo-achilis.
4. **Movement:**
 - Toes: as neurological exam.
 - Ankle:
 - Dorsi flexion (15°).
 - Planter flexion (45°).
 - Subtalar joint:
 - Inversion (20°).
 - Eversion (10°).
5. **Deformities :**
 - Hallux valgus in H.F. & ballet dancing.
 - Hammer toe.
 - Talipes equinus.
 - Pes cavus.

Knee

1. **Skin:**
2. **Muscles.:** wasting by guttering or tape meter
3. **Swelling:** (swelling)

Effusion			
Front		back	
Inspection	Palpation	Inspection	Palpation
Lost depression around patella	1. Bulge test for mild effusion. 2. Patellar tap for moderate effusion. 3. Fluctuation for tense effusion. 4. Joint line: feel the tibial & femoral joint.	Popliteal fossa for Baker cyst	Emiflexed knee & palms of both hands on popliteal fossa

- Synovium. : by sliding test there is gritty sensation.
- S.C. nodules.

4. Movement:

➤ ROM.:

- Flexion (distance between heel & buttock).
- Extension (compare with other leg).

5. Crepitation.**6. Deformities.**

- Genu valgus.
- Genu varus.
- Flexion deformity.

7. Tenderness: articular & extra articular.**8. Testes for collateral ligaments.****Hips****1. Skin:****2. Muscles :****3. Movement: gait**

- Flexion.

4. Crepitus:**5. Deformity:****6. Tests:** sciatic nerve pressure (midway between greater trochanter & ischial tubrosity)**Spine****1. Deformity :**

- Localized angular deformity in pott's, fracture, malignant deposition.
- Kyphosis: increased with aging.
- Scoliosis:
 - On both standing & sitting (structural)
 - On standing & decreased by sitting (positional) as in unequal leg length.
- Lordosis:
 - Cervical : lost (pain) \ \ increased (ankylosing spondilitis).
 - Lumbar : lost (A.S.\disc\old age)\ \ increased (myopathy).
- Movement: active & if failed passive.
- Tenderness: fist \ hemmer \ 2 thumb pressure \ index between spines.

Cervical**Mid. Cervical:**

- Flexion (chin to chest).
- Extention (occiput to wall).

Atlanto-occipital: head nodding.**Atlanto-axial:** lateral rotation.

Thoraco-lumber

Flexion:

- Finger to floor test (with extended LL it's > 10 cm)
- Shouber's test.

Extention:

- backward movement.

N.B.:

- Painful flexion & extention = A.S. - O.A. - discs.
- Limited flexion & extention = A.S.

Lateral tilting:

Special joints

T.M.J.: In Rheumatoid arthritis.

- Open, close, protrude, retract.
- Palpate for tenderness & crepitus.

Sacro iliac joint: (patient is supine)

1. Springing of the pelvis :

- Abduct pelvis by both hands from SIS leads to pain.

2. Faber test = patric test

- Knee flexion + hip abduction + external rotation. (to put heel of the patient on the opposite knee & press on flexed leg down to the bed leads to contralateral pain.

3. Knee flexion + hip adduction + internal rotation leads to ipsilateral pain. Patient prone

- Press on the sacrum .
- Press on the iliac crest to compress the pelvis.

- C/P, Inv., TTT & Rh. A.
- C/P, Inv., TTT & SLE.
- C/P, Inv., TTT & Scleroderma



See written notes

Rheumatoid arthritis

Criteria for Rheumatoid Arthritis : (4 / 7)

1. **Morning Steffness > 1. hr.**
2. **Arthitis of > 3 joints (soft tissue swelling).**
3. **Swelling of PIP / MCP or writ joint.**
4. **Symmetrical swelling of joints.**
5. **S.C. nodules.**
6. **Rheumatic factor:** positive
7. **X- ray:** erosion + bony decalcification articular or peri articular.

N.B.

- Criteria 1,2,3 & 4 must be present for at least 6 weeks.

S.L.E.

Criteria of diagnosis (2 rash - 2 serology - 7 Ps)

1. Malar flush (**rash**).
2. Discoid **rash**.
3. **P**hoto sensitivity.
4. **P**ainless oral ulcers.
5. **P**ancytopenia.
6. **P**roteinuria or cast.
7. **P**elurisy or pericarditis.
8. **P**sycohosis or seizures.
9. **P**eripheral joint arthritis (2 or more).
10. Positive ANA antibody.
11. Immunological investigations: positive anti DNA ab or anti-sm ab.

The diagnosis of S.L.E. is strongly suggested, when a person has 4 or more of these criteria

- **Investigation of Rh. A. & S.L.E. → See written notse.**
- **TTT of Rh. A. & S.L.E. → See written notse.**

Diagnosis

- A case of poly arthritis with deformities & systemic manifestations, for D.D. most propably **Rh. arthritis**.

Rheumatoid factor (Rf)

Ig M, IgG or IgA against Fc portion of IgG

- Transient production of RF is an essential part of the body's normal mechanism for removing immune complex.

BUT in Rhd. A. they show a much higher affinity and their production is persistent and occurs in joints.

RF is not diagnostic for RF, nor does its absence rule the disease out, but it is a useful predictor of diagnosis.

- Apersistent high titre of R.F. indicates active erosive disease.
- R.F. + citrullinated cyclic peptide Abs → Together are more specific (ccp)

Condition in which R.F. is found in the serum

R.F. (70%)	Systemic sclerosis (30 %)	T.B
SLE (25 %)	Hepatitis	Elderly
Sjogren's (90 %)	IMN	Relatives of patient with RA
	Sarcoidosis	

Methods to detect R.F.

1. **Rose- walar**: usages of sheep RBCs
2. **Bentonite**: usages of clay
3. **Latex**: usages of synthetic material

Antinuclear antibodies (ANAs)

- Detected by indirect immune fluorescent staining of fresh frozen section of rat liver or kidney.
- Used in screening of SLE. But may be present in a low Titre in some diseases as follow:

SLE (95 %)	Autoimmune hepatitis (100 %)
S.S. (70 %)	Drug- induce lupus (95 %)
Sjogren's (80 %)	M.G. (50 %)
Rhd. A. (30 %)	Normal population (8 %)

Anti extractable nuclear Aq antibodies:-

1. **anti Ro** → SLE + Sjogren's S.
2. **anti la** → Sjogren's S.
3. **anti Sm** → SLE.
4. **anti U 1 RNP** → overlap syndrome.

Anti citrullinated protein antibodies

(ACPA)

Def.: Anti Ab. That is detected in cases of Rhd. A. during inf. Arginin protein enzymeticaly converted into citrullin (procast called citrullination)

The shape of this protein well significantly altered.

So may be seen as a foreign prt. By immune system

So (ACPA) has proved to be powerful Biomarker for diagnosis of Rhd. A. at very early stage.

Useful in follow up.

Scleroderma

Progressive systemic sclerosing (PSS)

Def: Generalized disorder of connective tissue characterized by fibrosis and degenerative changes in **blood vessels**, **visceral** organ and **skin**

- No inflammation
- Clinical hallmarks of PSS are tight skin and Raynaud's phenomenon
- Diagnosis made on clinical grounds (no specific Abs 100%)

Incidence:

- F:M = 3 - 4 : 1 ☺
- Incidence peaks in 5th and 6th decade (may be earlier)
- Associated with HLA DRI, DR3, DR5
- May associated environmental factors

Pathogenesis:

- Fibroses and degenerative changes in **skin and viscera**
- No inflammation - collagen deposition → atrophy and fibrosis.
- Fibroses and degenerative changes in **blood vessels**
- Decreased vascular luminal size: **intimal** proliferation and **medial** degeneration progressive obliteration of vessel lumen → secondary fibrosis of tissue.

Clinical picture

skin

- Early: edema.
- Later: induration (hardening) of the skin with loss of its elasticity.
- Skin hyper/ hypo-pigmentation, ulceration & calcification.
- **Raynaud's phenomenon:** initial complaint in 70 %, all patients eventually develop it during the course of their diseases.

G.I.T.

- Esophagus: **dysphagia**, reflux. (80 % of cases).
- Small intestine: malabsorption syndrome.
- Large intestine: constipation.

Renal

- Vasculitis of renal blood vessels: hypertension & CRF.
- **Scleroderma renal crisis**: it's a life-threatening condition manifested by sudden onset of malignant hypertension & ARF.

Cardiac

- Cardiomyopathy.
- Pericarditis & pericardial effusion.
- Ischemic heart disease.
- Heart block: fibrosis of the conducting pathways.

Chest

- Interstitial pulmonary fibrosis.
- Pulmonary hypertension.
- Aspiration pneumonia: due to esophageal reflux.

C.N.S

- Headache and stroke during hypertensive renal crisis.

Musculoskeletal

- Arthralgia & rarely arthritis.
- Myopathy.

Variants (types) of scleroderma:**1. Diffuse scleroderma:**

- Diffuse skin hardening involving trunk & proximal limbs as well as face & distal limbs.
- Severe internal organs damage and is generally more life threatening.

2. Limited scleroderma: (CREST syndrome)

- Skin hardening is limited to the fingers, face and feet.
- Internal organ involvement is not severe, much better prognosis.
- CREST (**C**alcinosis, **R**aynaud's, **E**sophageal dysmotility, **S**clerodactyly, **T**elangiectasia).

3. Localized scleroderma: (scleroderma without internal organ involvement).

- Morphoea: local patches of scleroderma.

4. Systemic sclerosis *sin* (without) scleroderma

- Internal organ involvement without skin manifestations (rare).

5. Overlap syndrome: scleroderma associated with other autoimmune disease.

Overlap syndrome(mixed connective tissue disease): this syndrome involves mixed features of at least 2 connective tissue disorders (S.L.E., Scleroderma, Polymyositis).

Investigations

1. ↑ ESR & CRP.
2. ANA: positive in 90 % of patients.
3. RF: positive in 20 % of patients.
4. Anti Scl- 70: 30 % of diffuse scleroderma.
5. **Anti - centromere:** 70 % of limited scleroderma.
6. Biopsy.

Because not all people with scleroderma have these antibodies and because not all people with antibodies have scleroderma, lab test results alone cannot confirm the diagnosis.

Treatment

General: Avoidance of exposure to cold

Specific:

- Penicillamine of little value
- Steroids: in early phases
- Others: methotrexate/cyclosporine

Symptomatic treatment

- GERD: proton pump inhibitors
- Small bowel bacterial overgrowth: broad-spectrum antibiotics (tetracycline, metronidazole)
- Raynaud's: calcium channel blockers, peripheral vasodilators, local nitroglycerin cream, systemic PGE2 inhibitors
- Renal disease or HTN: ACE inhibitors
- Myositis or pericarditis: steroids.

Behcet's disease

An inflammatory disorders of an unknown aetiologic

Epidemiology:-

- Common in Turkey, Iran & Japan
- With HLA-B51

C/P:-

Diagnostic criteria:-

Recurrent oral ulcers and 2 of the following

- Genital ulcer
- Defined eye disease (Ant. Or post. Uveitis)
- Skin Lesion as erythema nodosum
- Positive pathergy test

Others:-

- Mono or oligo arthritis - C.N.S. (as M.S.)
- GIT manifestation
- Pulmonary lesion
- Renal lesion

N.B.

- *All manifestation are self limited except uveitis which may cause blindness*
- *Pathergy reaction:- is highly specific, skin injury by a needle prick for example ; leads to papule or pustule formation within 24-48 hrs.*

• TTT:-

- Steroid & immune suppressive for eye manifestation & neurological complication.
- Colchicin for Joint & skin manifestation.
- Topical steroid for oral & genital ulcers.